Left Ventricular Hypertrophy: Spotting the Signs Beyond Hypertension

57th Annual Primary Care Review
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Disclosures

- None

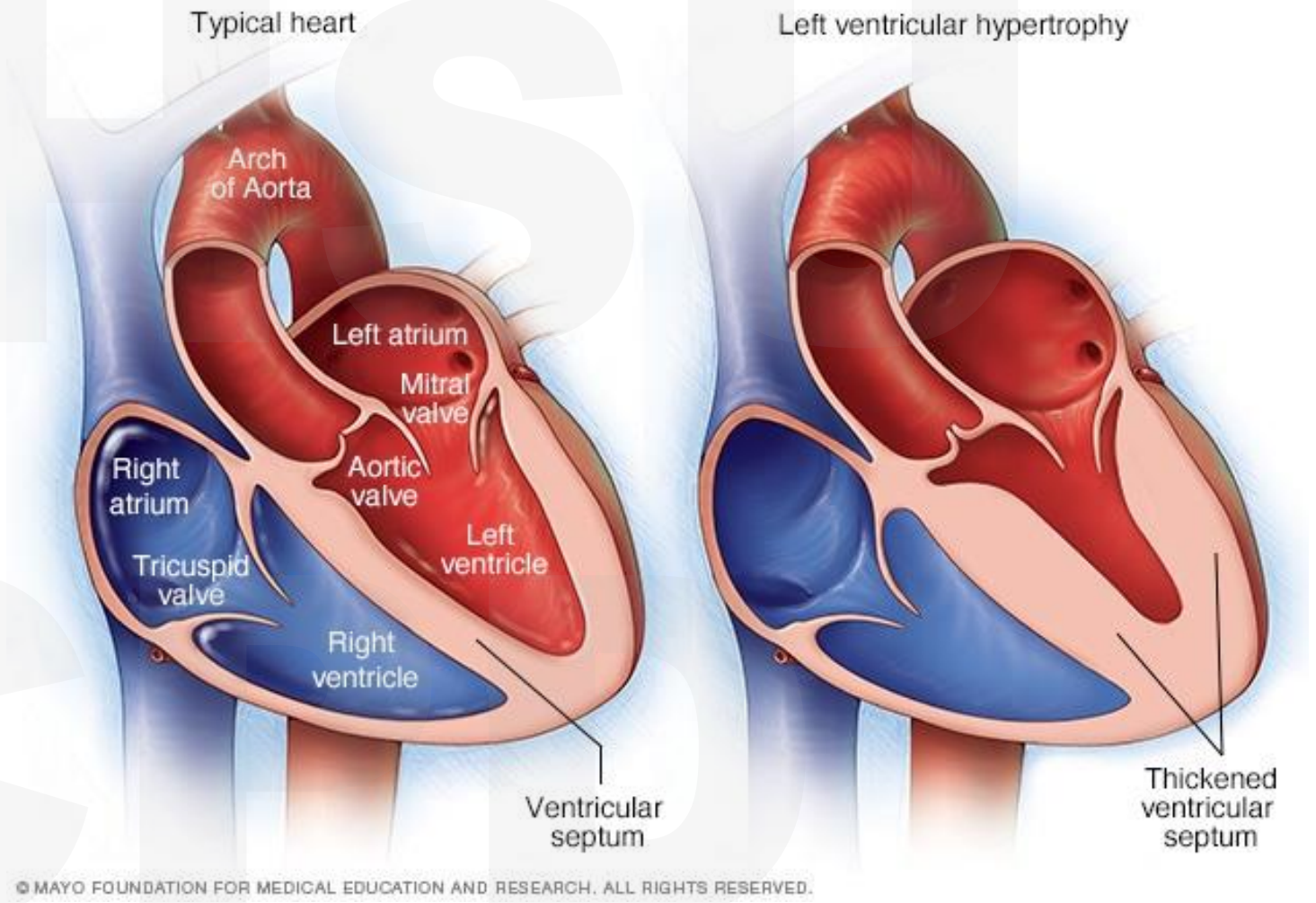
Objectives

1. Review common causes of LVH
2. Recognize Hypertrophic Cardiomyopathy (HCM) and Cardiac Amyloidosis (CA)
3. Laboratory and imaging evaluation for LVH phenotypes
4. Treatments for HCM and CA

What is left ventricular hypertrophy?

- ↑ wall thickness = ↑ mass

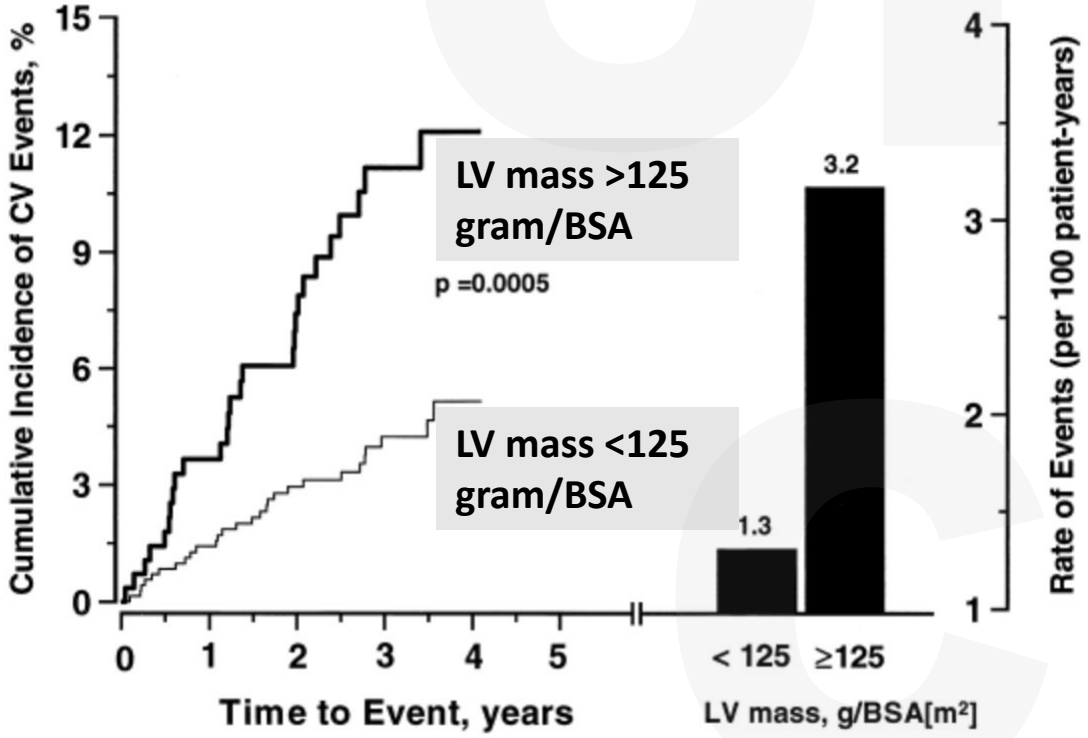
- Most common mechanism is cardiac myocyte hypertrophy from **HTN**



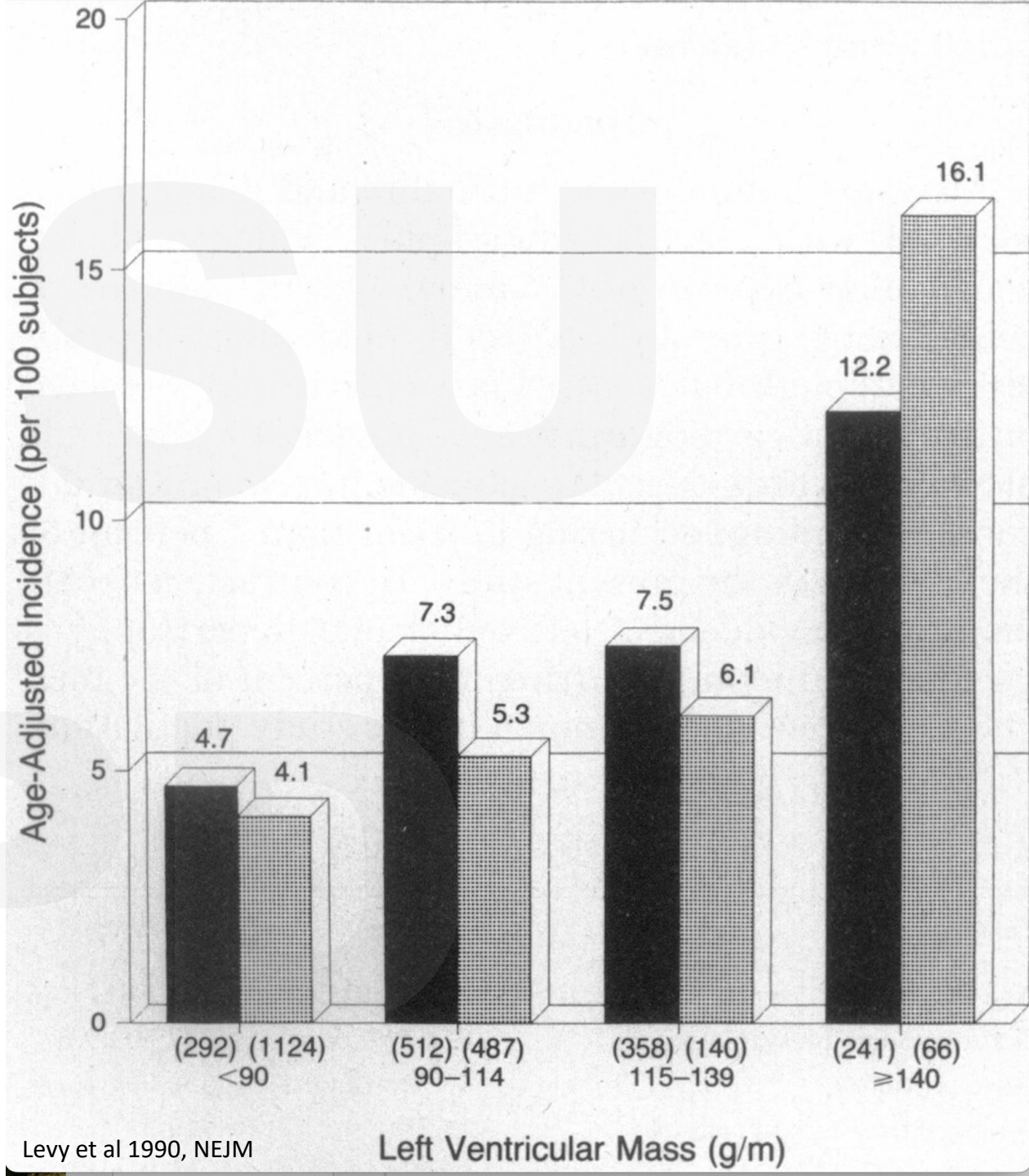
Which of the following is a common cause of LVH?

1. Aging
2. Aortic Stenosis 
3. Obesity
4. Ischemic heart disease

Why do we care about LVH?

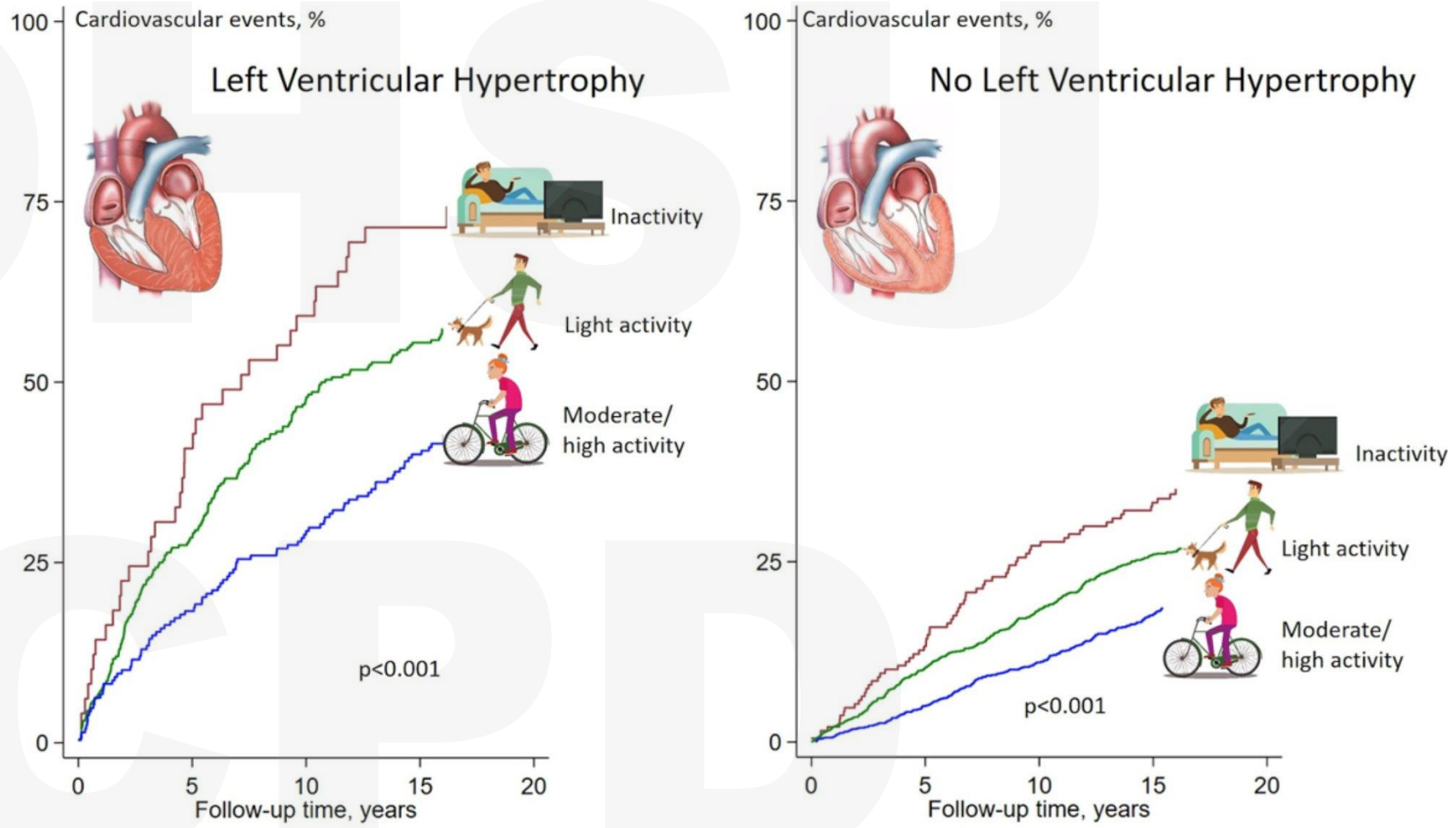


Verdecchia et al 2001, JACC



Levy et al 1990, NEJM

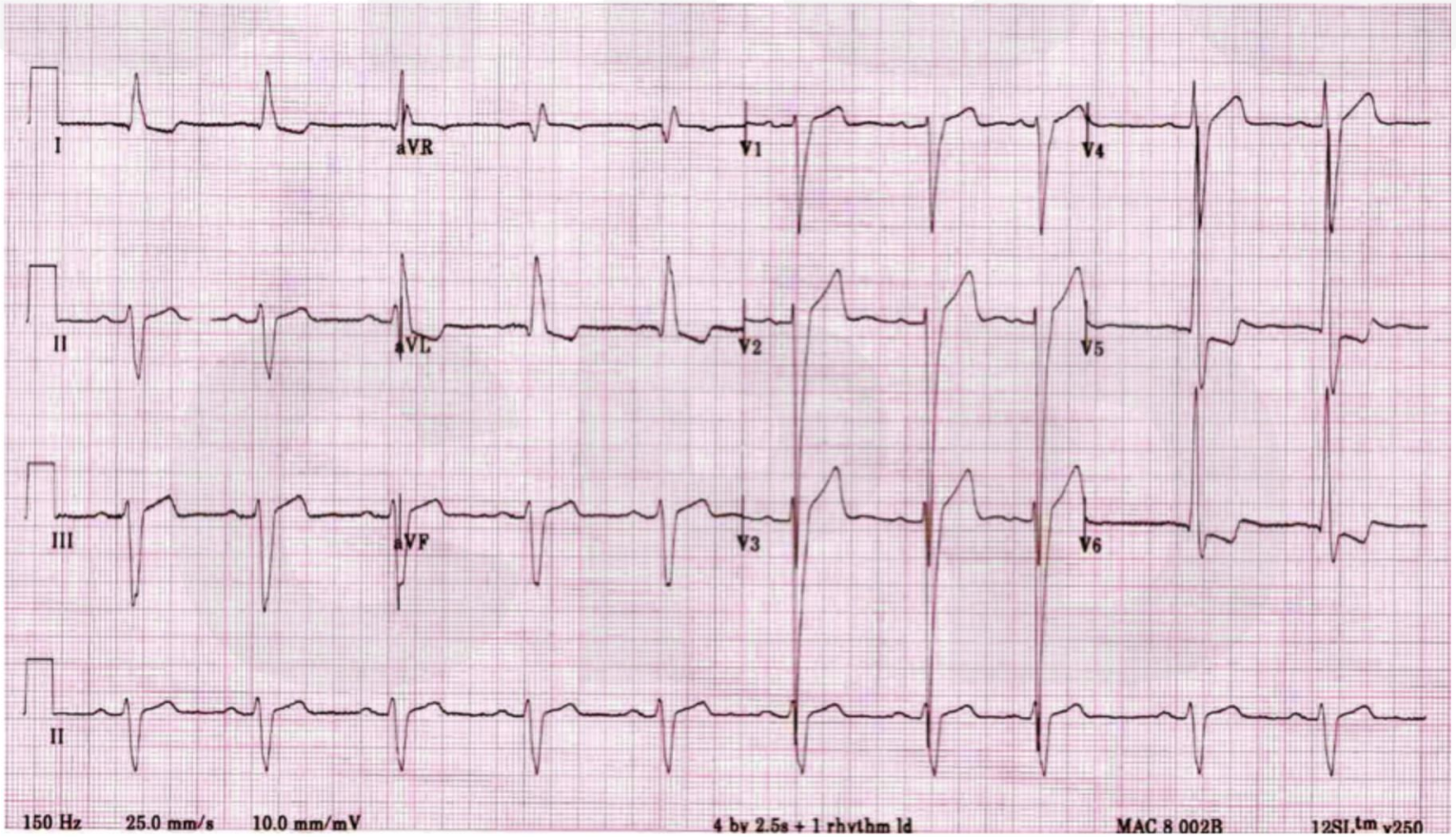
Even in active individuals LVH matters



Joseph et al 2020, Hypertension

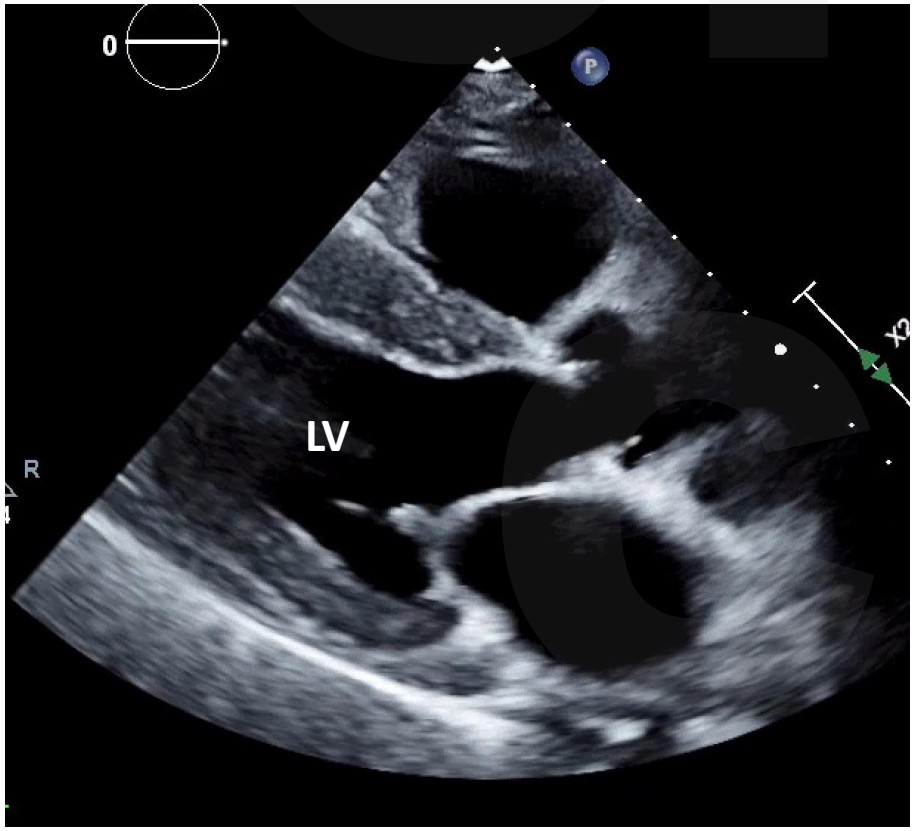
How do we detect LVH?

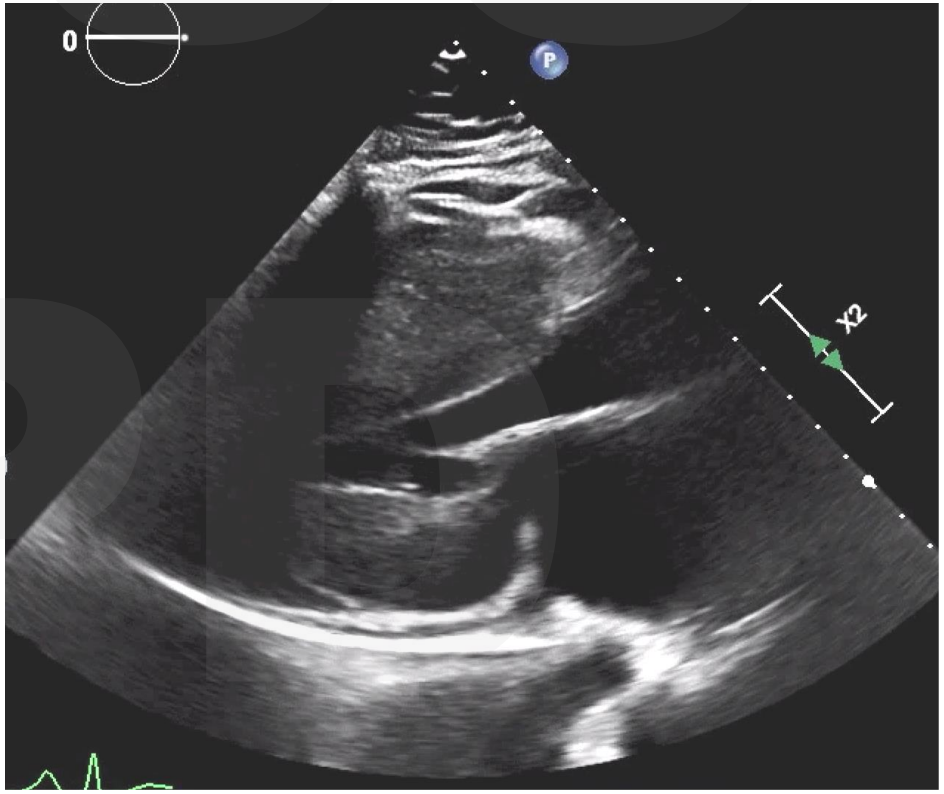
Electrocardiographic Criteria for Left Ventricular Hypertrophy	
Sokolow-Lyon Criteria	S V_1 + R $V_{5,6}$ > 35 mm OR R aVL > 11 mm
Cornell Criteria	R aVL + S V_3 > 28 mm for men OR > 20 mm for women



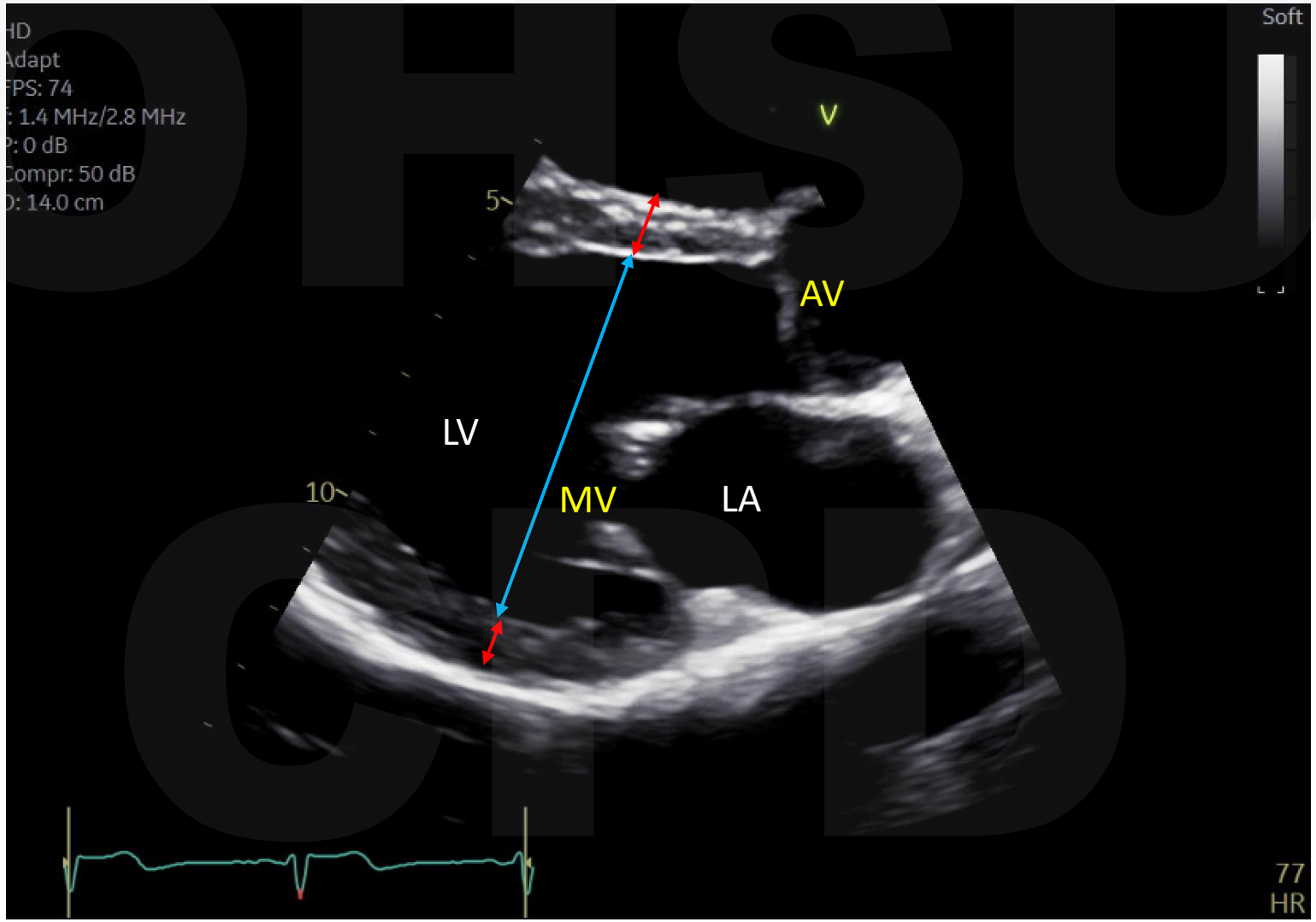
Echocardiographic findings of LVH

- Structural and morphologic information
- Hemodynamic information

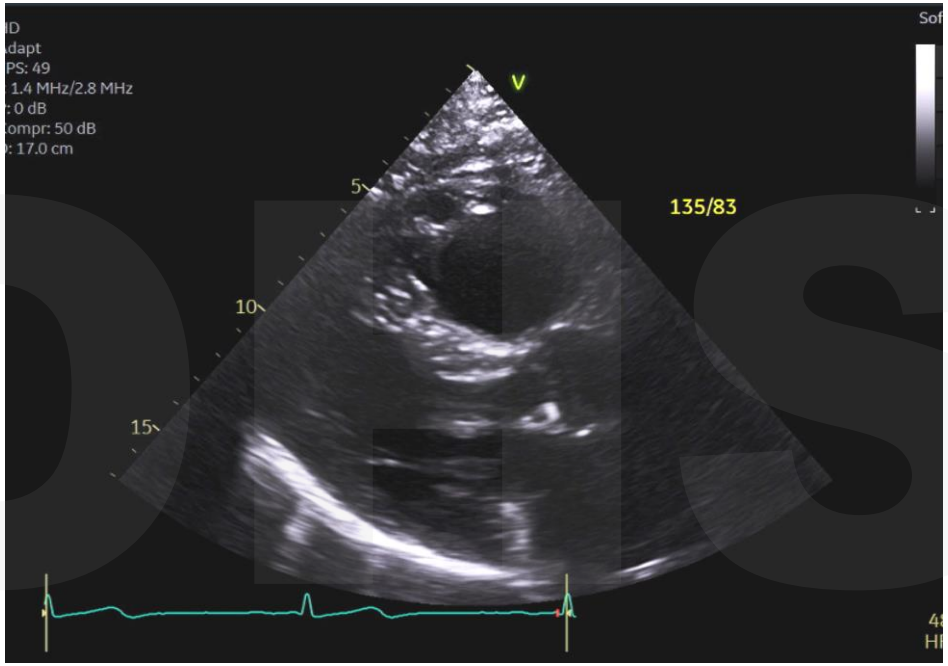


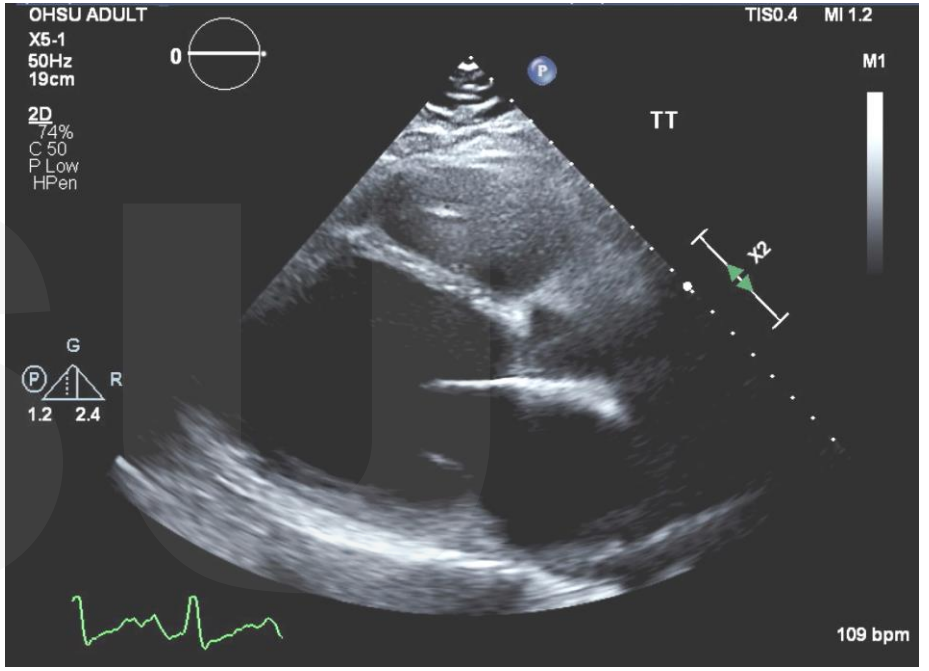


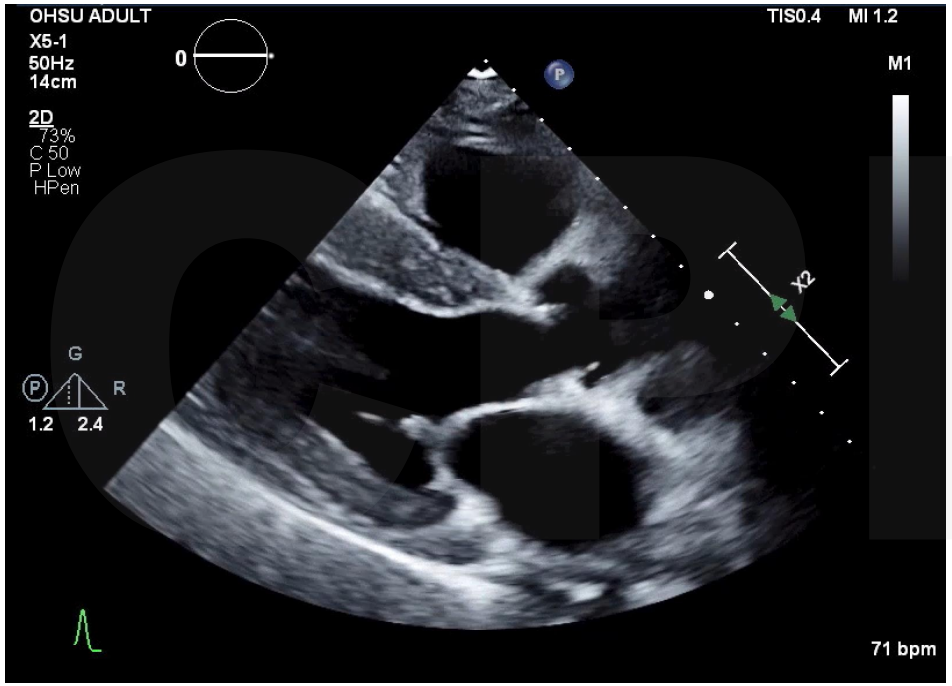
How is LVH measured?

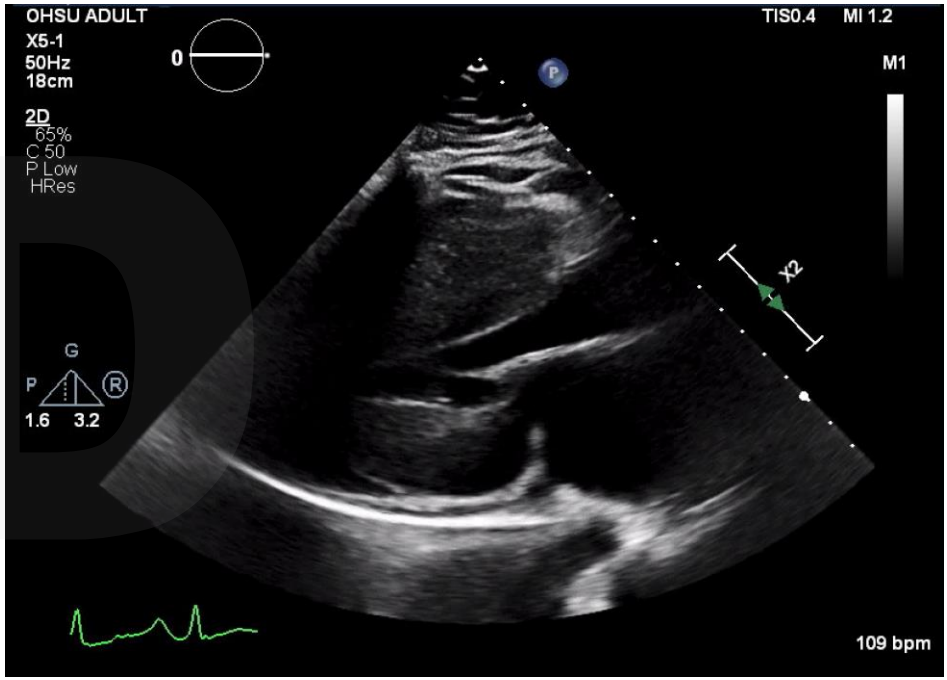


Not all LVH is the same









Etiology of LVH determines treatment

- Chronic hypertension – management of blood pressure
- Severe aortic stenosis – treat aortic stenosis
- Hypertrophic cardiomyopathy – cardiac myosin inhibitors
- Infiltrative cardiomyopathy – multiple therapies depending on etiology

LVH Cases

Case 1

HPI/CC: 65yo female with essential hypertension, T2DM, CKD III presents to clinic to establish care with chief complaint of shortness of breath.

Medications: insulin

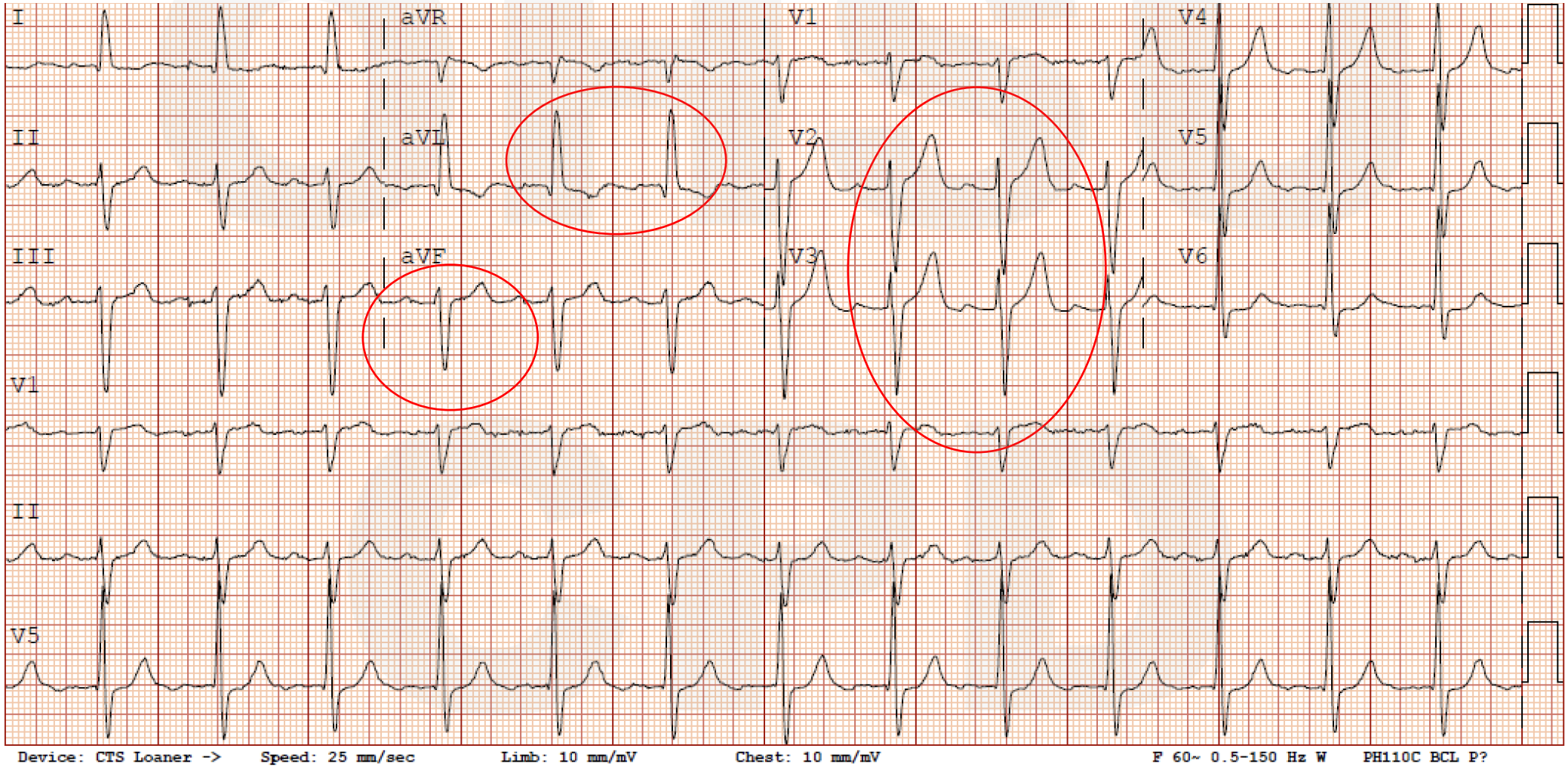
Allergies: None

Family history: father – T2DM, mother – MI age 67

Vitals: afebrile, BP 168/94, HR 60, 96% O2

Exam: RRR, no murmurs, JVP normal, trace peripheral edema

ECG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
Device: CTS Loaner ->
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
F 60~ 0.5-150 Hz W
PH110C BCL P?

Initial management

Baseline labs ordered, plan to start ARB for hypertension

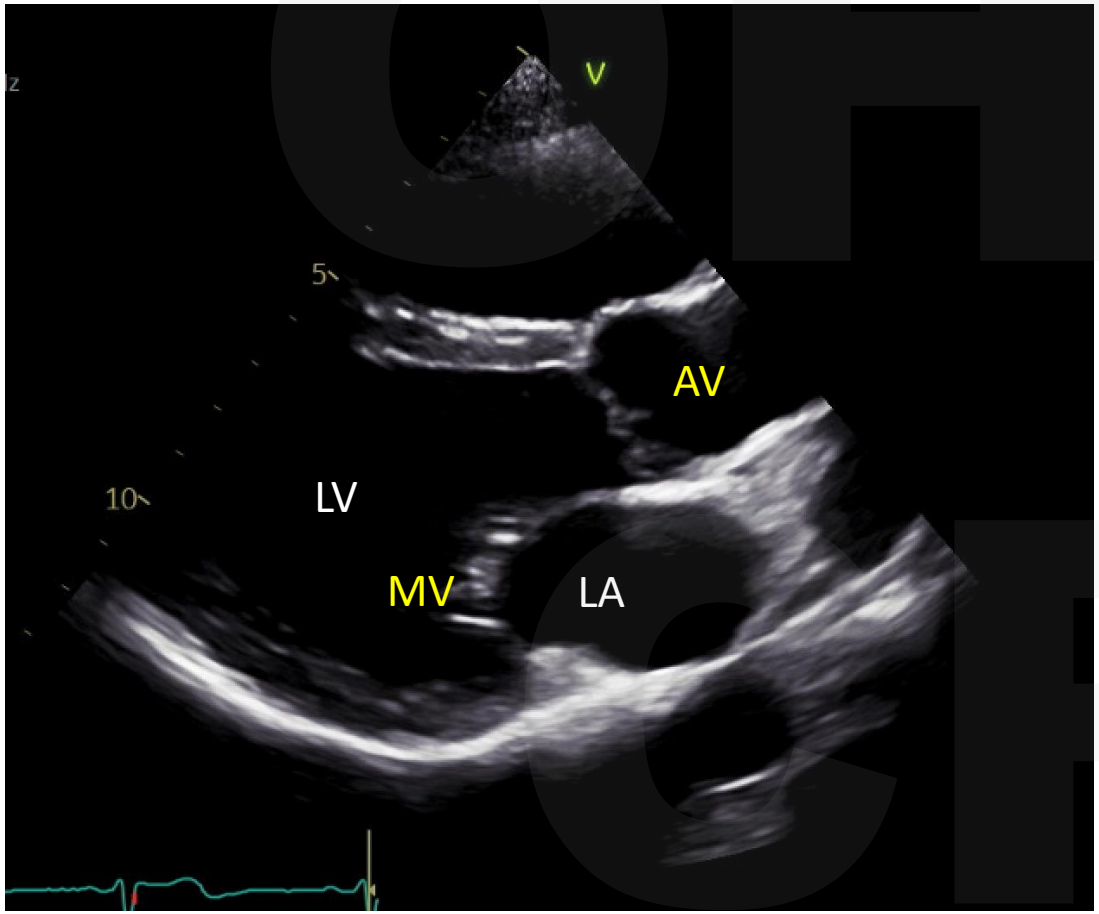
Ordered echocardiogram

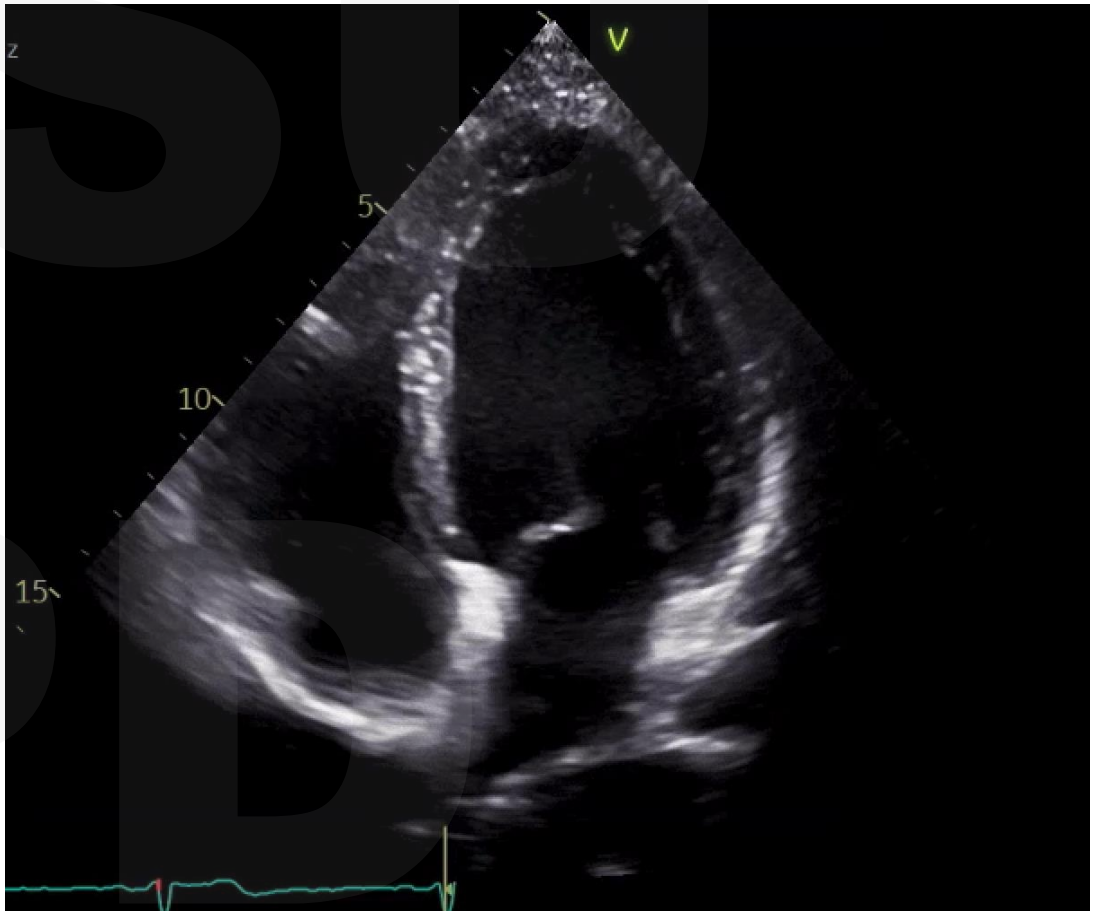
Echo report

Final Impressions:

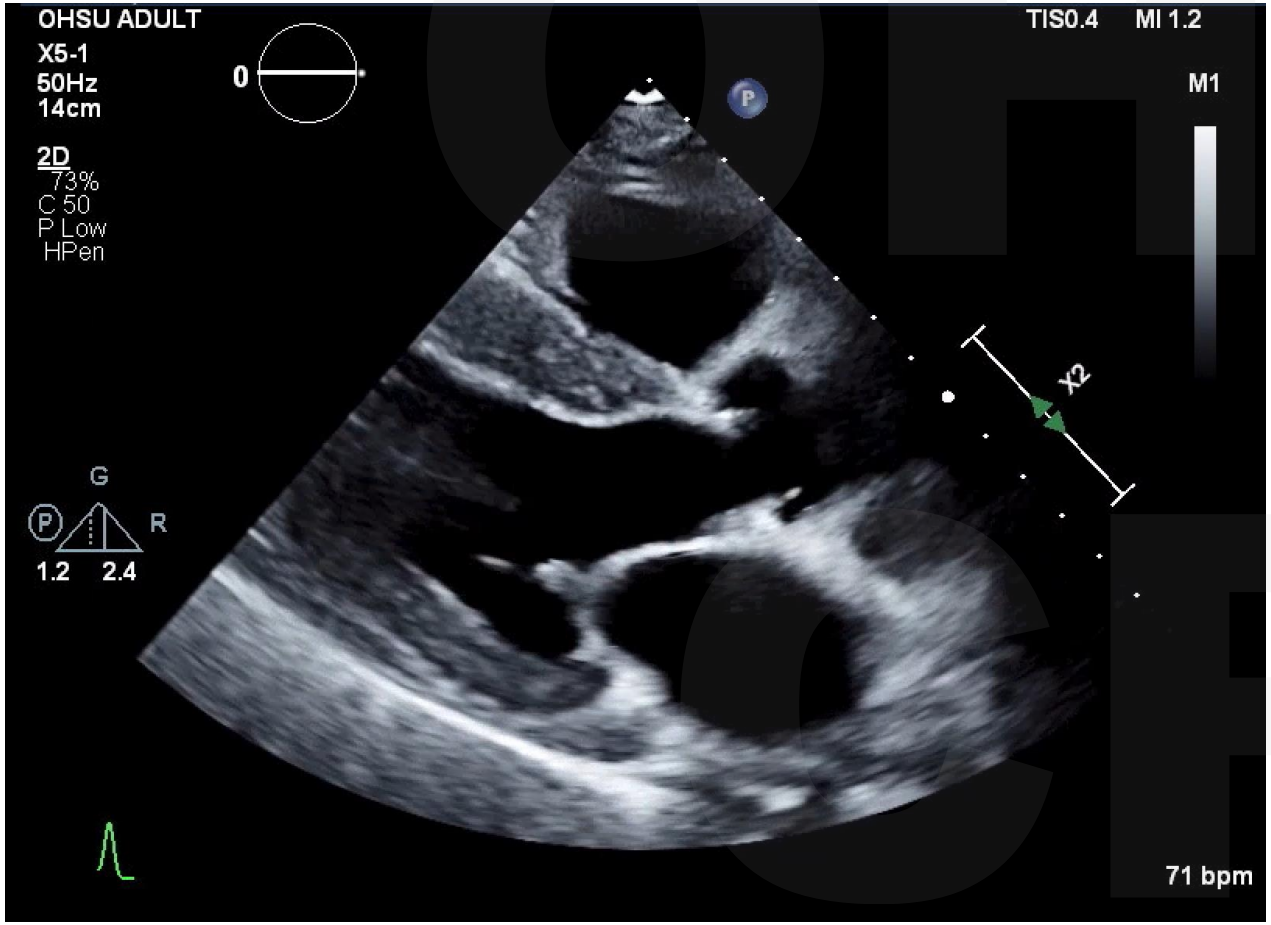
- The left ventricular size is normal
- The LV function normal
- There is moderate concentric left ventricular hypertrophy
- Right ventricular size, thickness and function are normal
- Mild biatrial enlargement

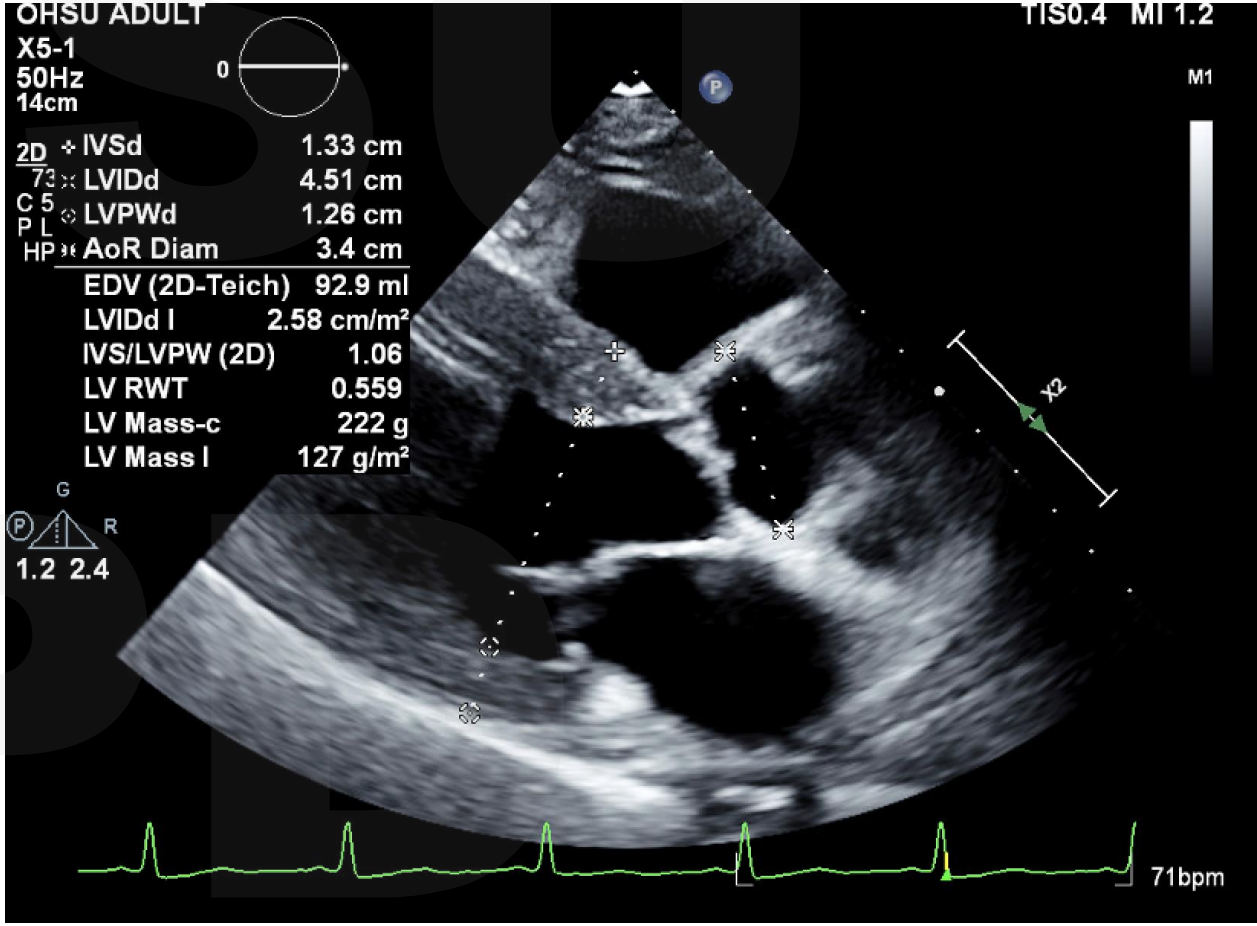
Normal Echo for reference



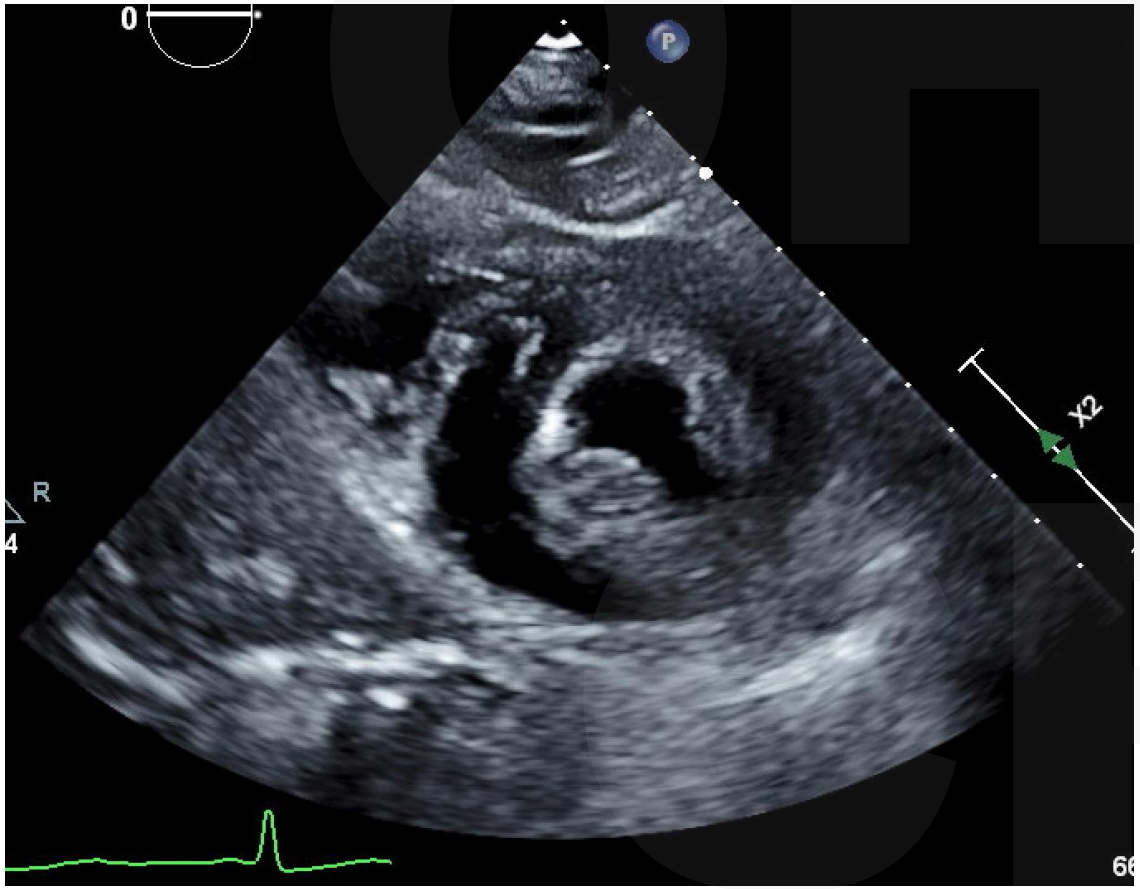


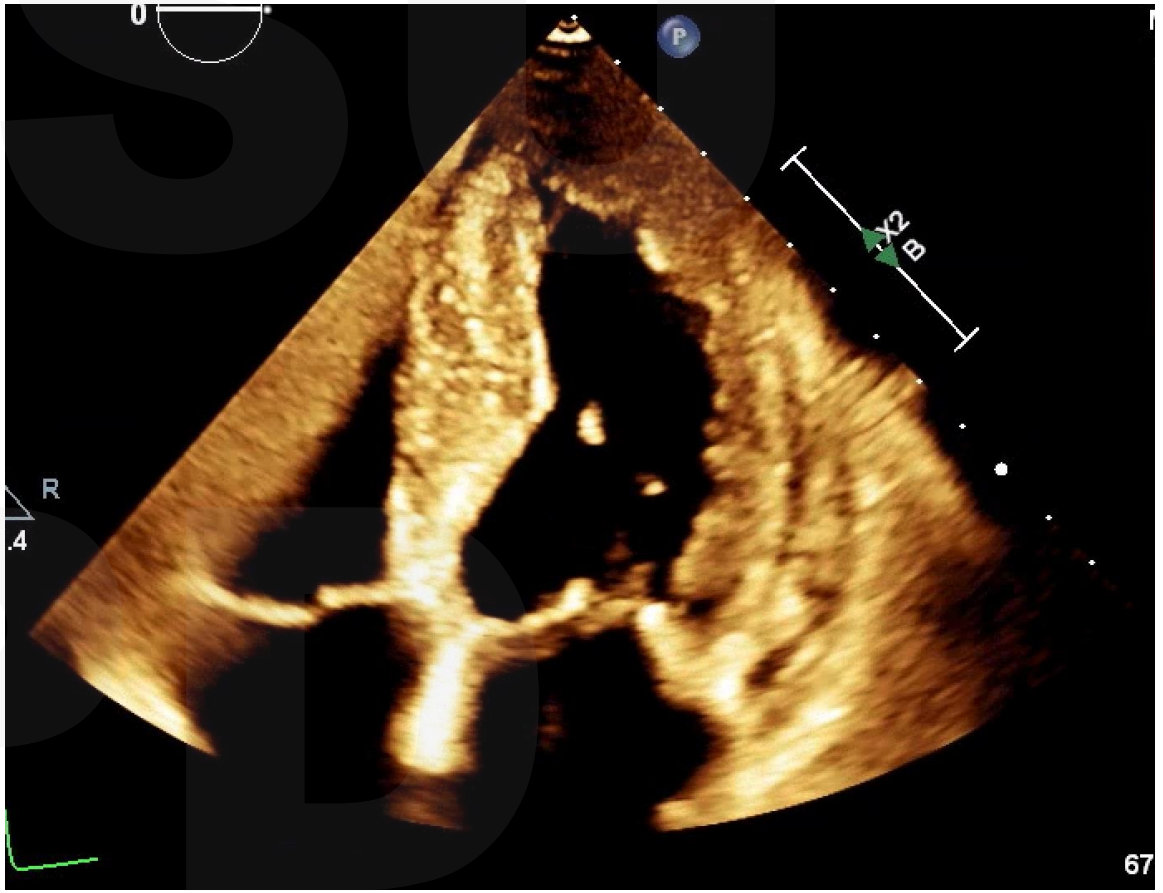
Echocardiogram





Echo continued



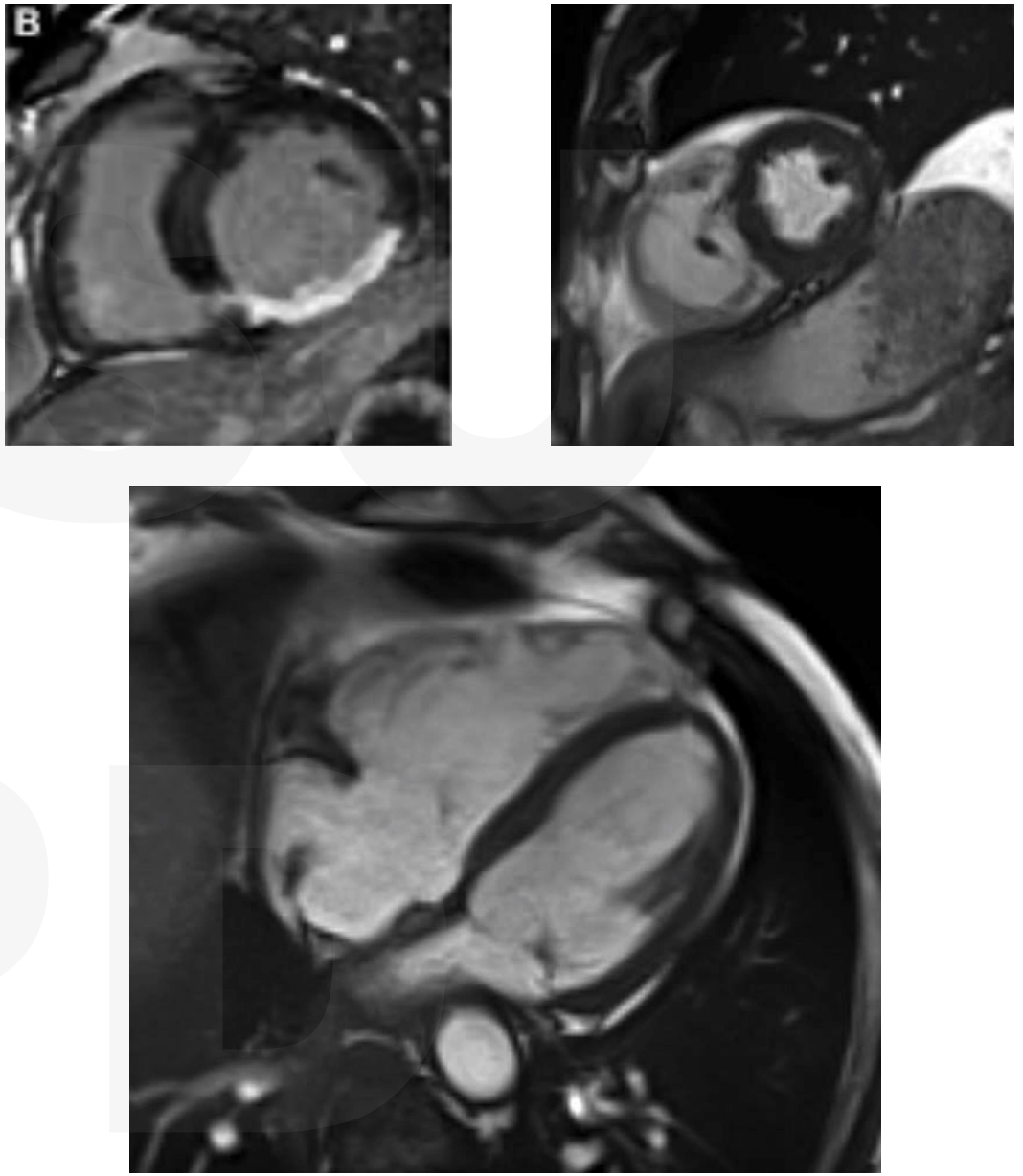


Follow-up visit

- Labs showed Cr 1.64, eGFR 42, K 4.1, NTproBNP 342
- BP still 160/92
- Suspect LVH 2/2 chronic hypertension, but no long term BP data to support
- What test can help confirm the diagnosis?
- What medical therapy to start?

Cardiac MRI

- Provides highly detailed and accurate structural analysis of the heart
- Able to assess hemodynamics: cardiac output, shunts, valvular regurgitation
- Late gadolinium enhancement



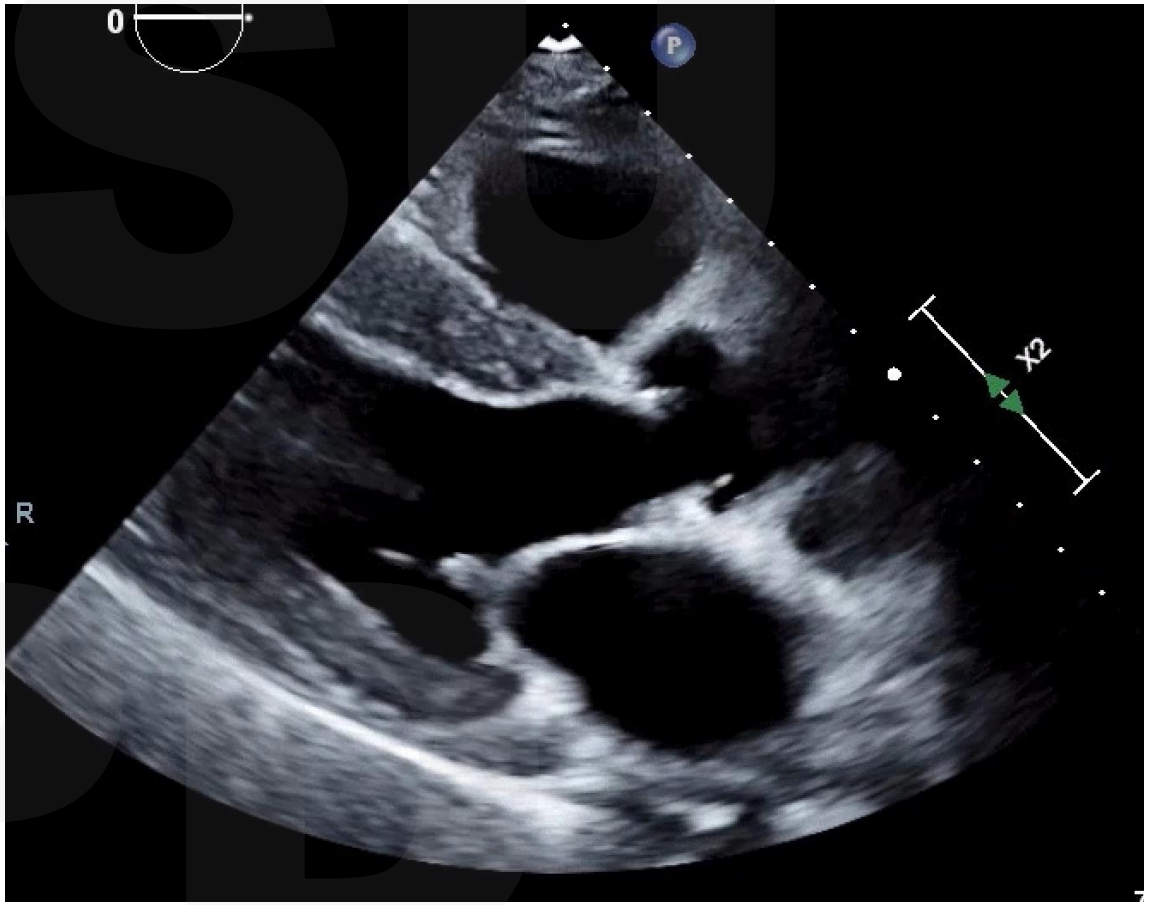
Medical therapy for LVH due to chronic hypertension

- Treatment is focused on controlling blood pressure
- Medications:
  - ARBs/ACEi and CCB have largest effect on reducing LV mass: 12-14%
  - Beta-blockers and diuretic had lower efficacy: 7-8%
  - LVH regression in 10-20%

Fagard et al, 2009, Circulation

LVH due to chronic hypertension

- Mild to moderate concentric LVH on echo
- Chronic hypertension is critical to diagnosis
- No additional evaluation if etiology is clear
- Treatment is focused on reducing blood pressure
- Regression will occur in some and is associated with reduction in cardiovascular events
- Consider cardiac MRI for LVH without clear HTN or aortic stenosis



Case 2

HPI/CC: 75yo male with essential hypertension (well controlled), T2DM, CKD III, peripheral neuropathy, presents to clinic to establish care with chief complaint of shortness of breath and mild lower extremity edema.

Medications: insulin

Allergies: None

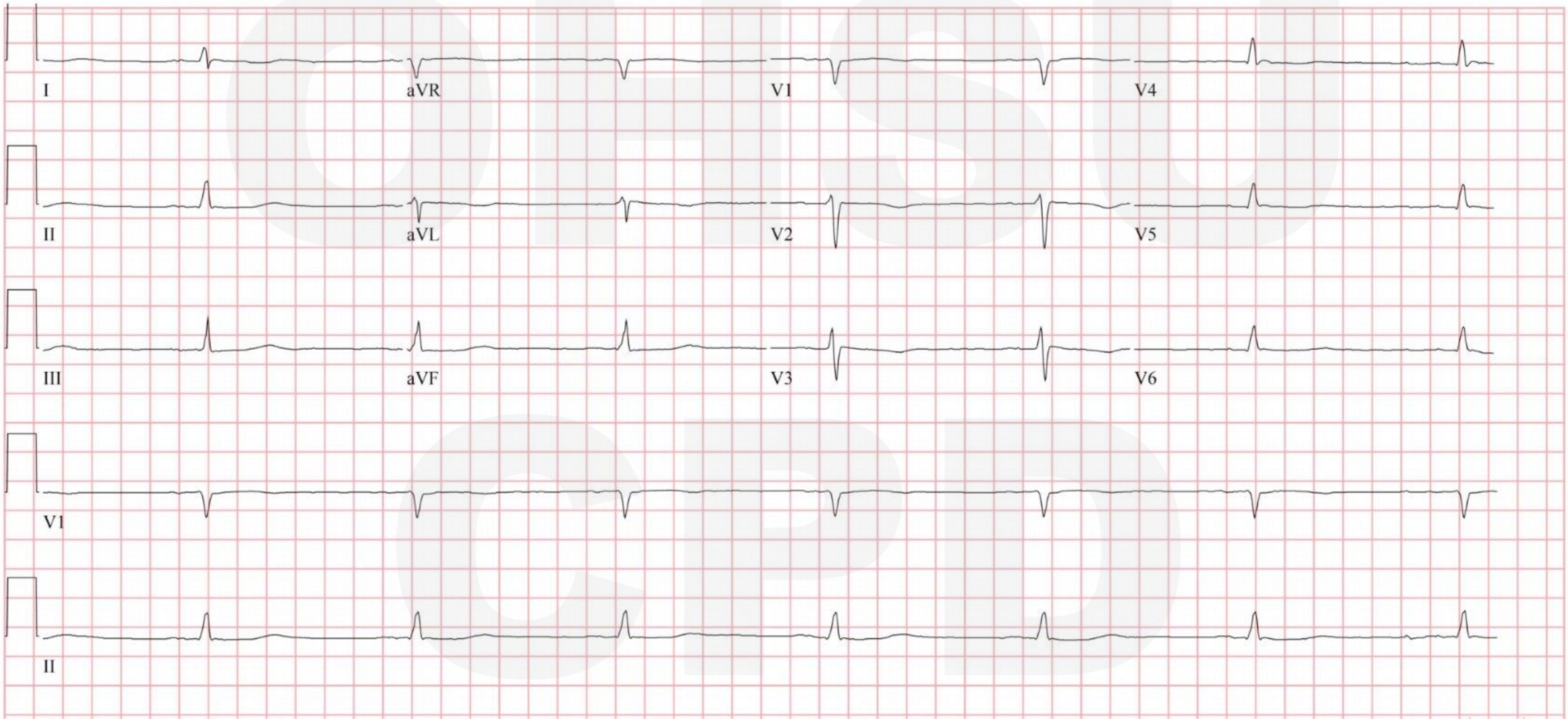
Family history: father – T2DM, mother – MI age 67

Surgical history: bilateral carpal tunnel surgery 15 years ago

Vitals: afebrile, BP 130/75, HR 60, 96% O2

Exam: RRR, no murmurs, JVP normal, trace peripheral edema

ECG

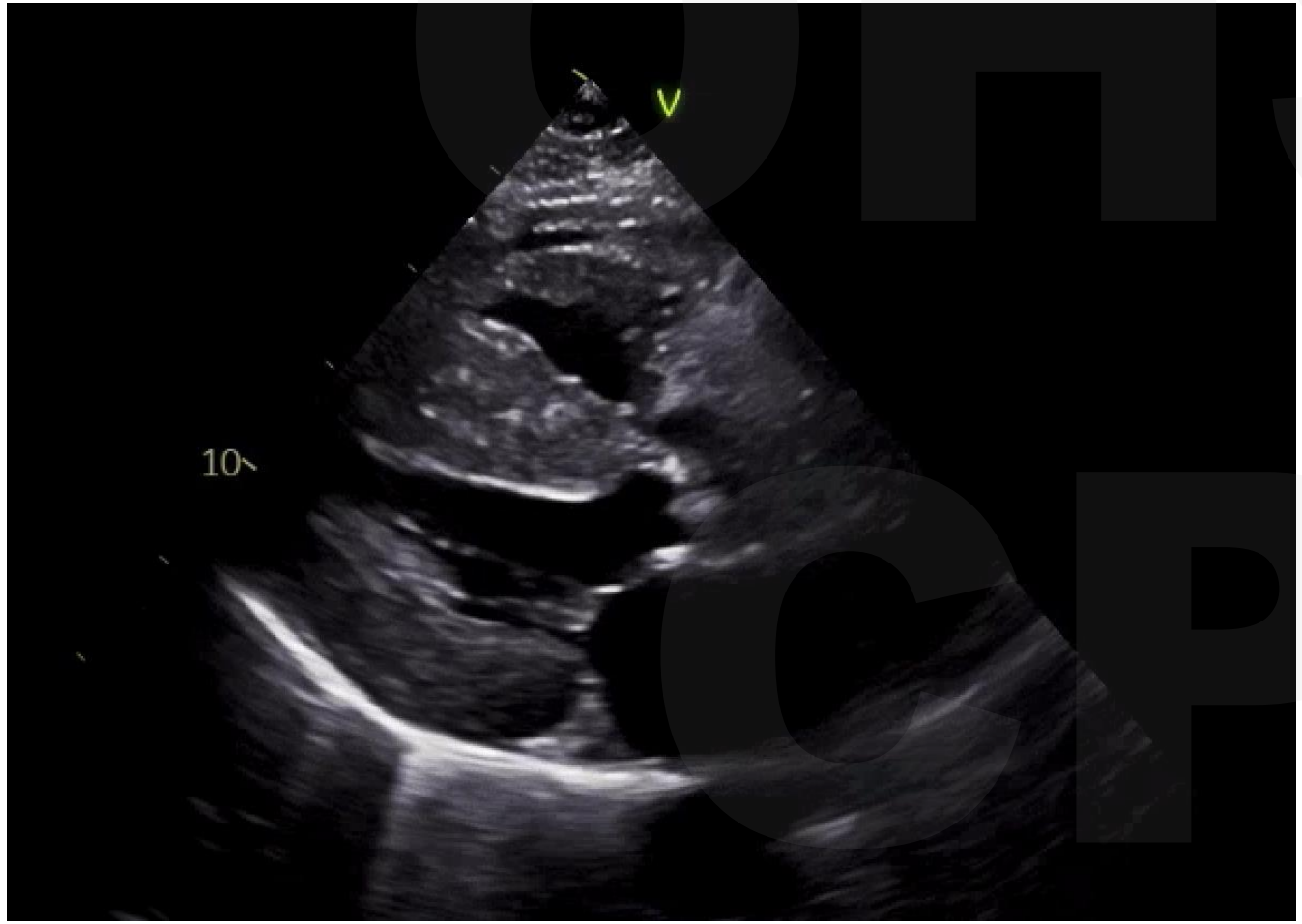


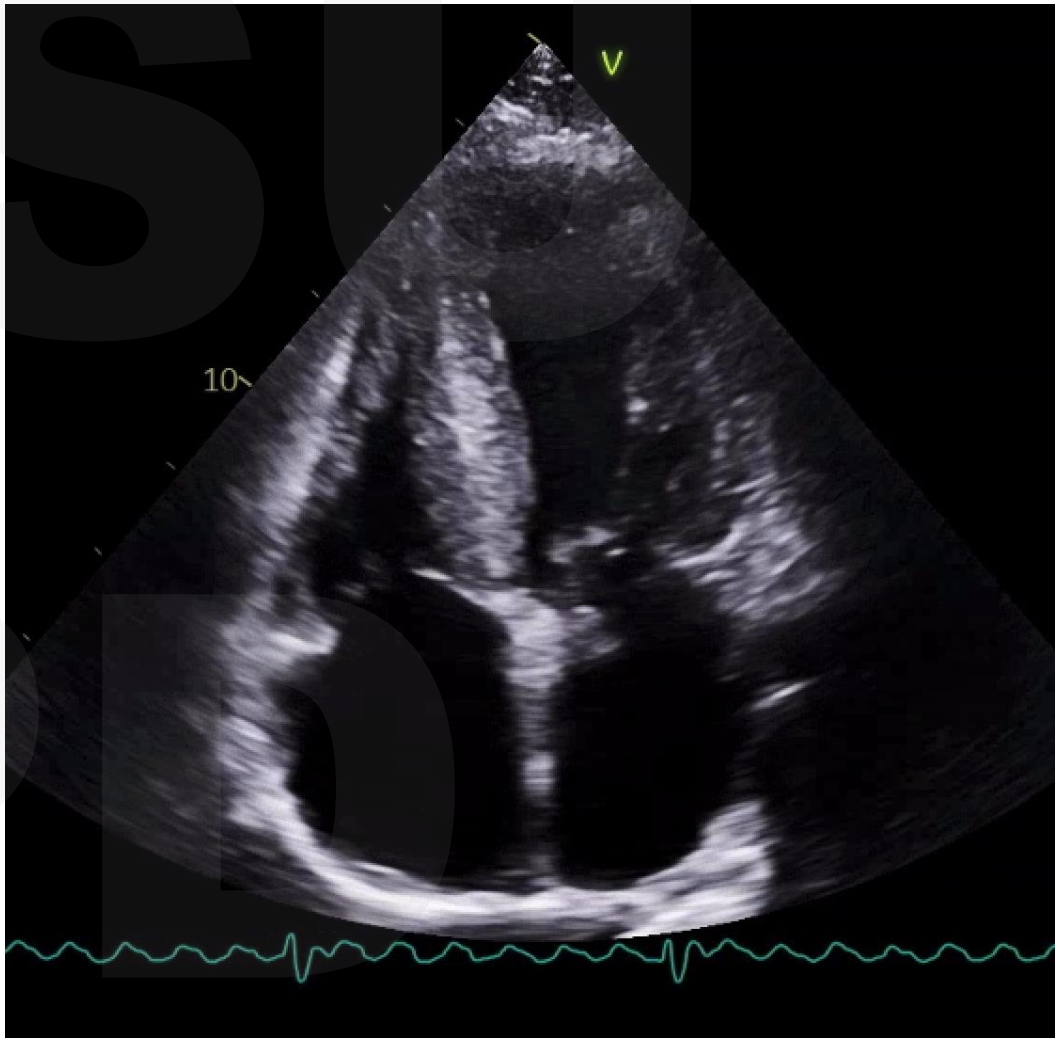
Echo report

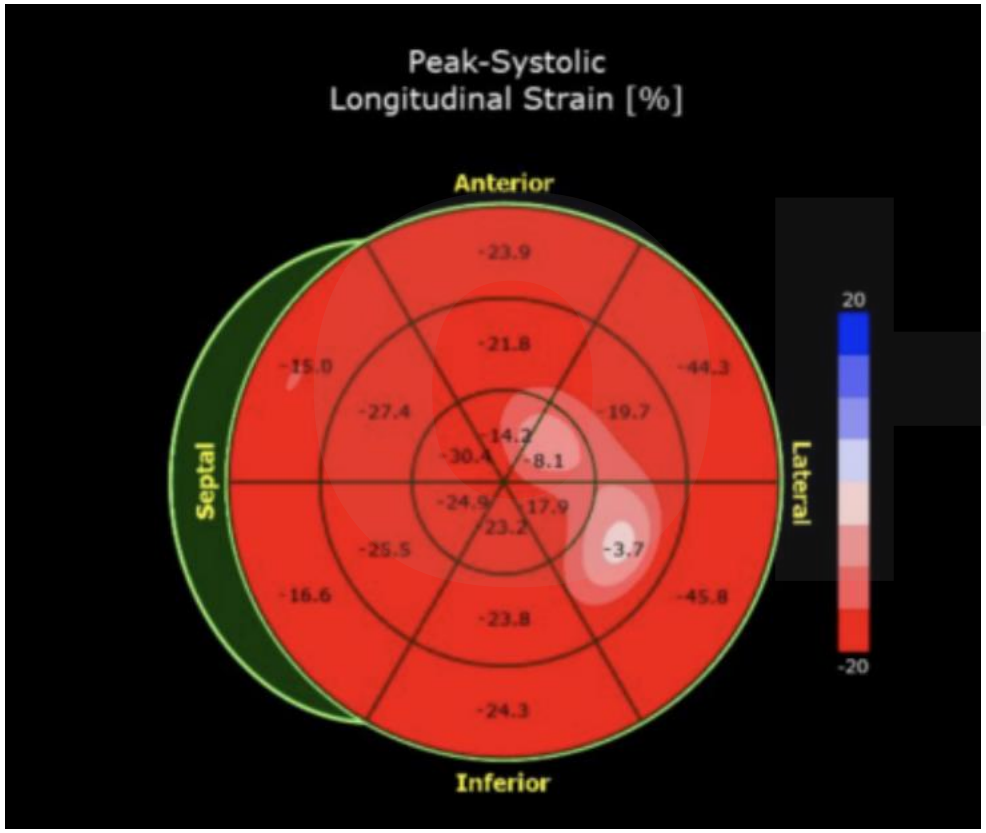
Final Impressions:

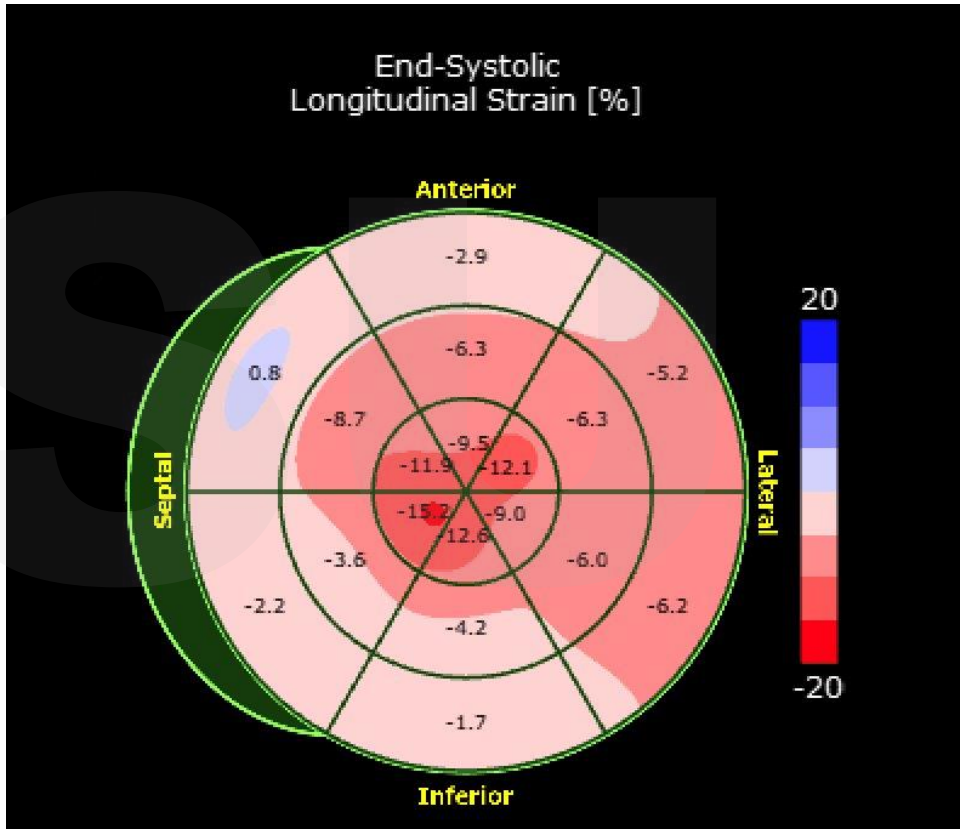
- The left ventricular size is normal
- The LV function is normal
- There is severe concentric left ventricular hypertrophy
- Visually estimated LV EF is 50-55%
- The aortic valve is trileaflet and mildly calcified
- Compared to prior exam, there are no significant changes

Echocardiogram









LV Strain Pattern

- Software that uses speckle tracking to assess systolic function
- Amyloidosis can have characteristic apical sparing strain pattern
- Sensitivity: 50-60%
- Specificity: 80-90%

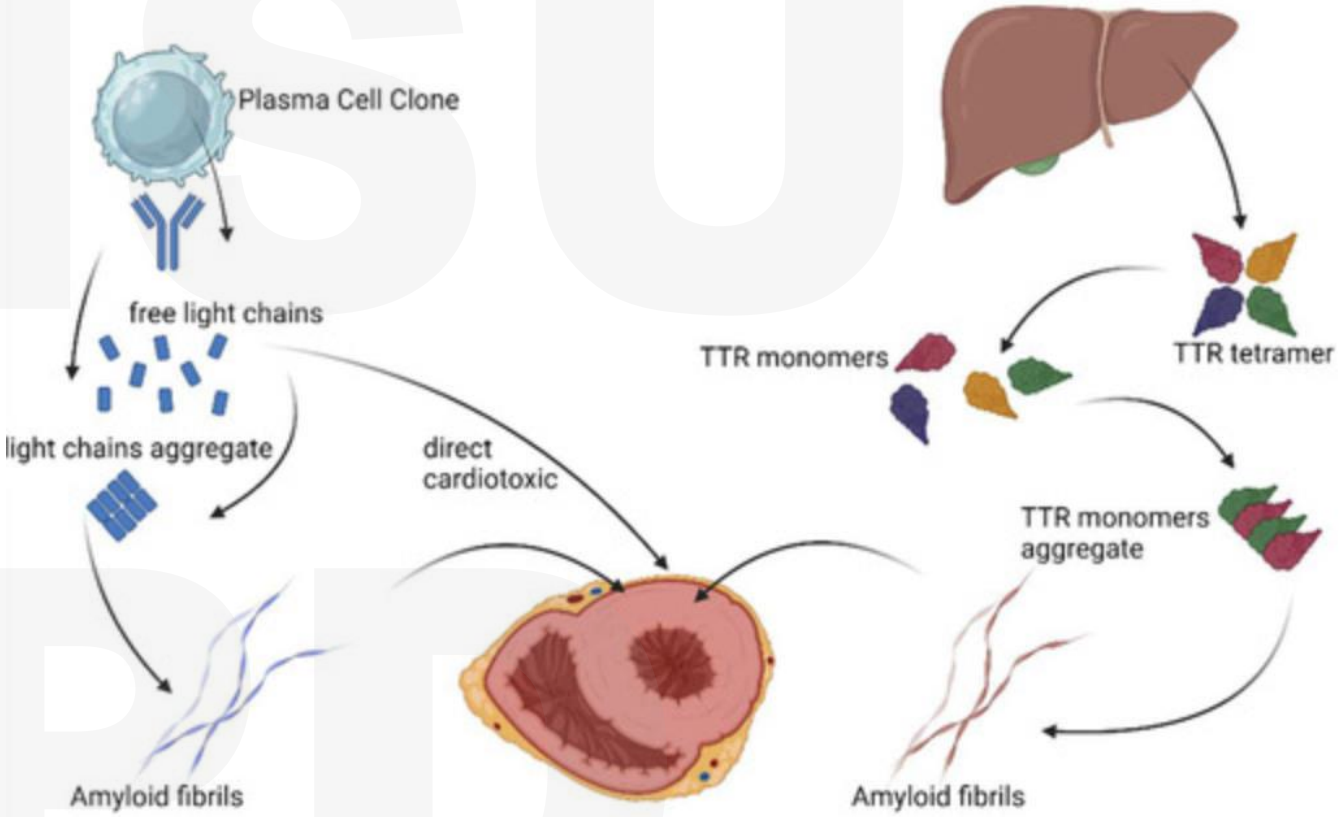
Khan et al, 2025, EHJ

Cardiac Amyloidosis

- Systemic disease resulting in deposition of amyloid fibrils in tissues leading to end organ damage

- Two forms of cardiac amyloidosis
  - Transthyretin amyloidosis (ATTR-CM)
  - Light chain amyloidosis (AL amyloid)

- ATTR-CM is more prevalent
- AL amyloid is rare but high mortality



Laboratory evaluation of cardiac amyloid

CBC, CMP, NTproBNP, troponin

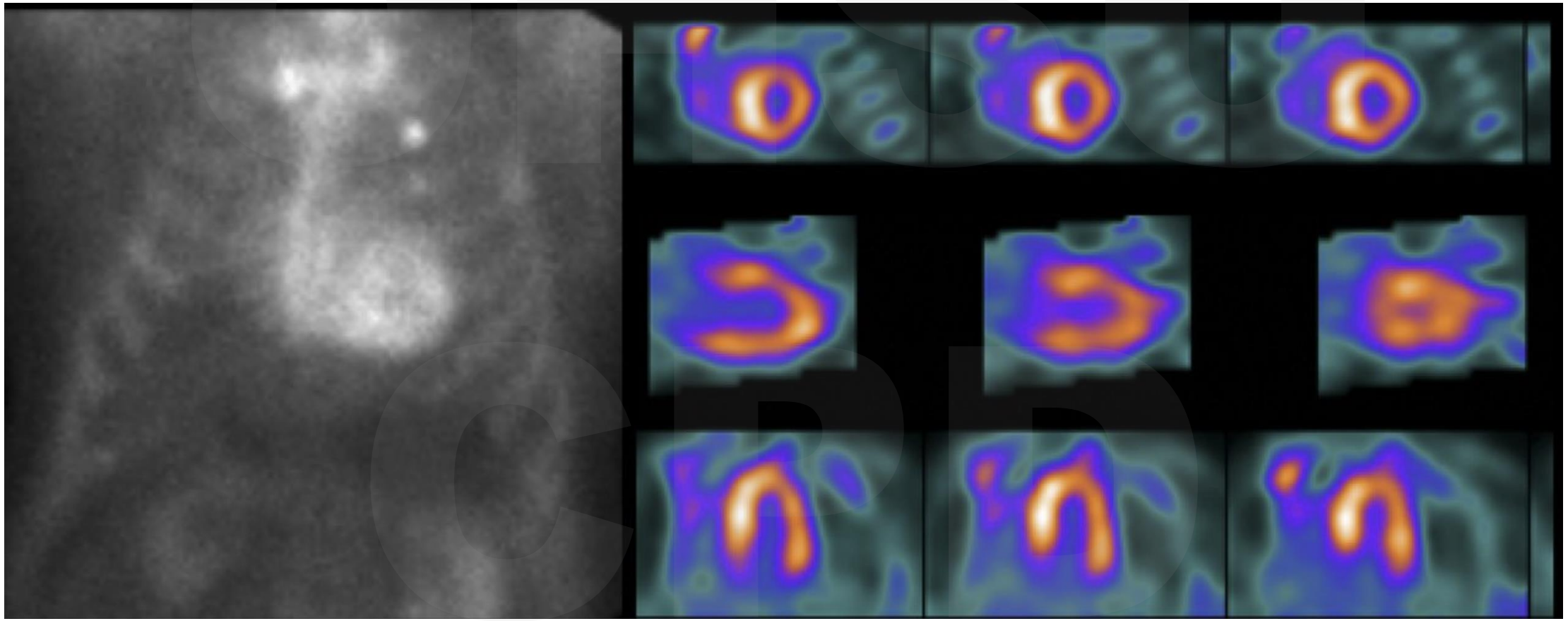
Serum free light chain ratio w/ immunofixation

SPEP and UPEP

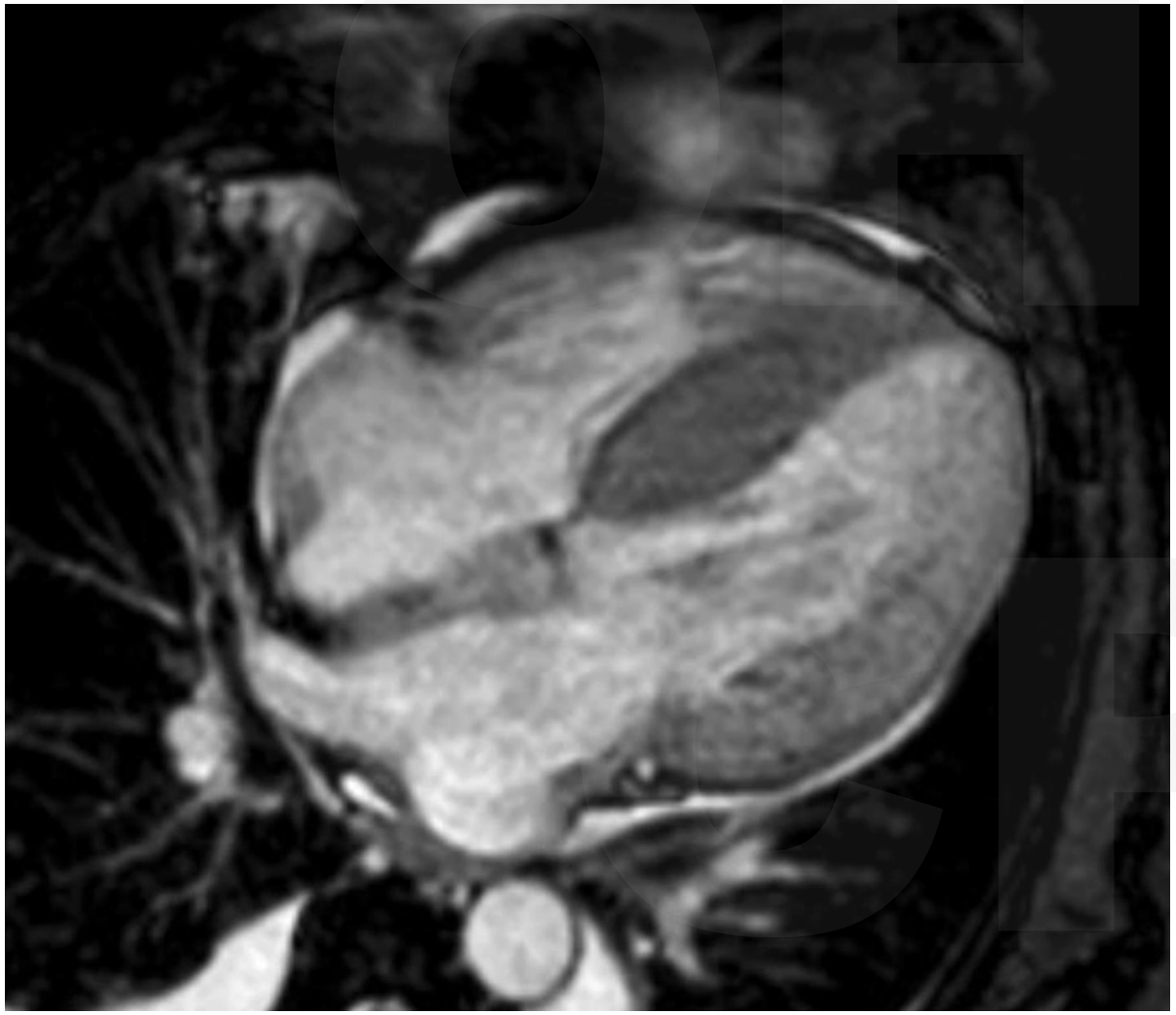
Lab findings concerning for AL amyloid

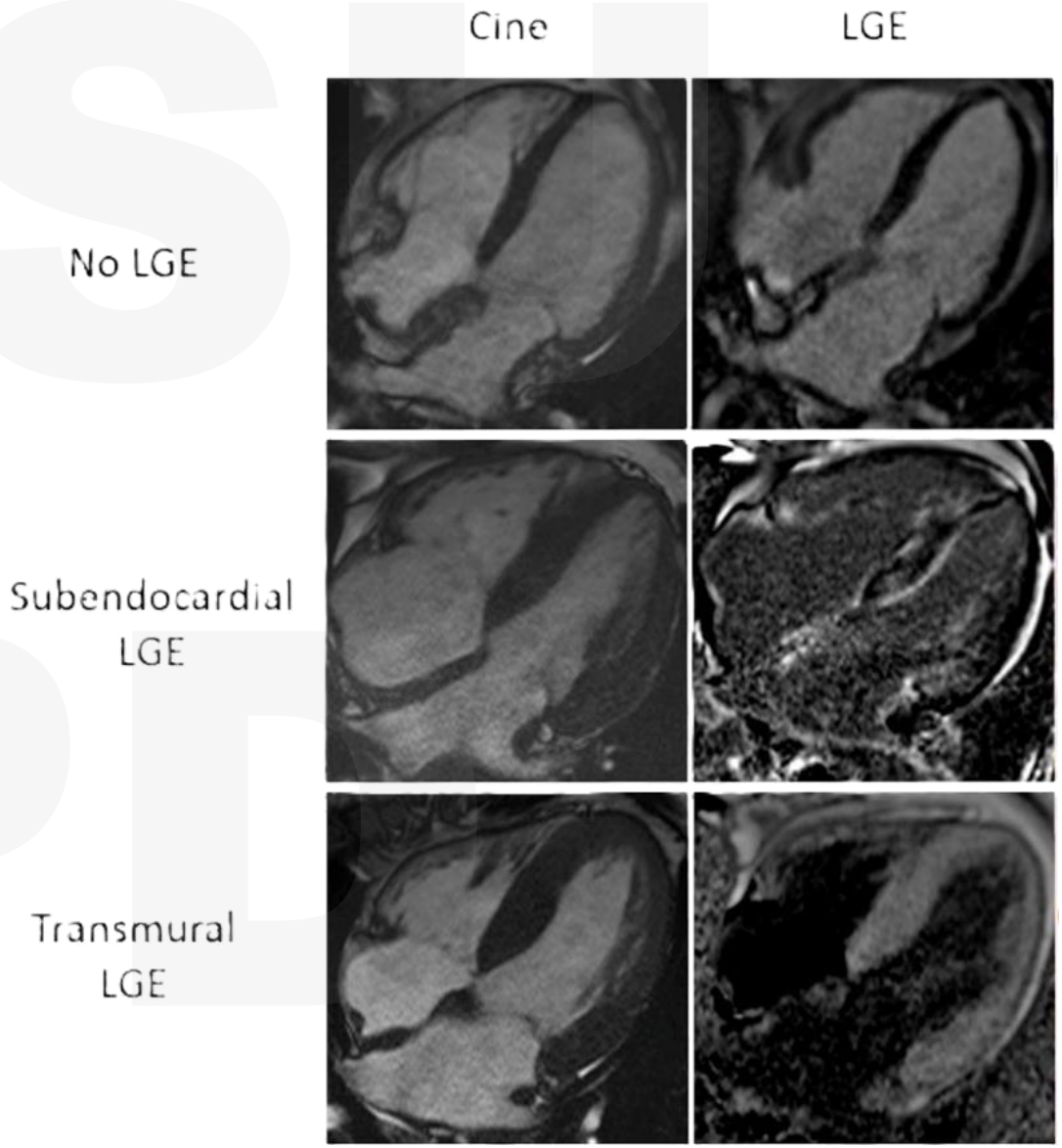
- Serum kappa:lambda ratio <0.26 or >1.65 w/ monoclonal protein
- M-spike on SPEP or UPEP

Multimodality imaging in cardiac amyloidosis



Cardiac MRI in ATTR-CM





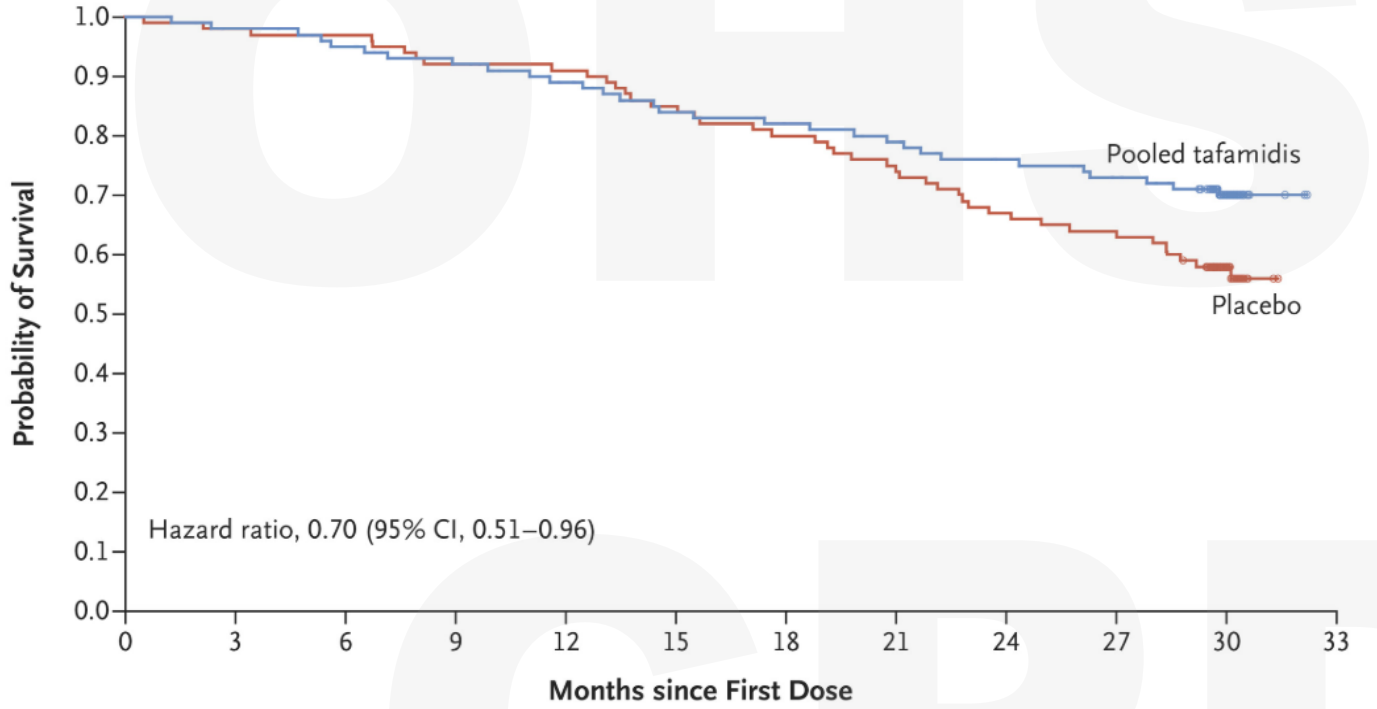
Which of the following is an established therapy for cardiac amyloidosis?

- Metoprolol
- Entresto
- Spironolactone
- Tafamidis ★

The NEW ENGLAND JOURNAL of MEDICINE

ATTR-ACT Study

B Analysis of All-Cause Mortality



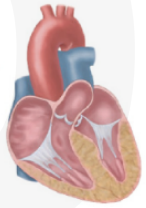
No. at Risk (cumulative no. of events)												
Pooled tafamidis	264 (0)	259 (5)	252 (12)	244 (20)	235 (29)	222 (42)	216 (48)	209 (55)	200 (64)	193 (71)	99 (78)	0 (78)
Placebo	177 (0)	173 (4)	171 (6)	163 (14)	161 (16)	150 (27)	141 (36)	131 (46)	118 (59)	113 (64)	51 (75)	0 (76)

So we can diagnose ATTR-CM, can we treat it?

Maurer et al, 2018, NEJM

Tafamidis prevents halts disease progression but does not reverse amyloid deposition

624 patients with transthyretin amyloid cardiomyopathy (ATTR-CM) treated with tafamidis at 5 U.S. amyloidosis centers (2018-2021)

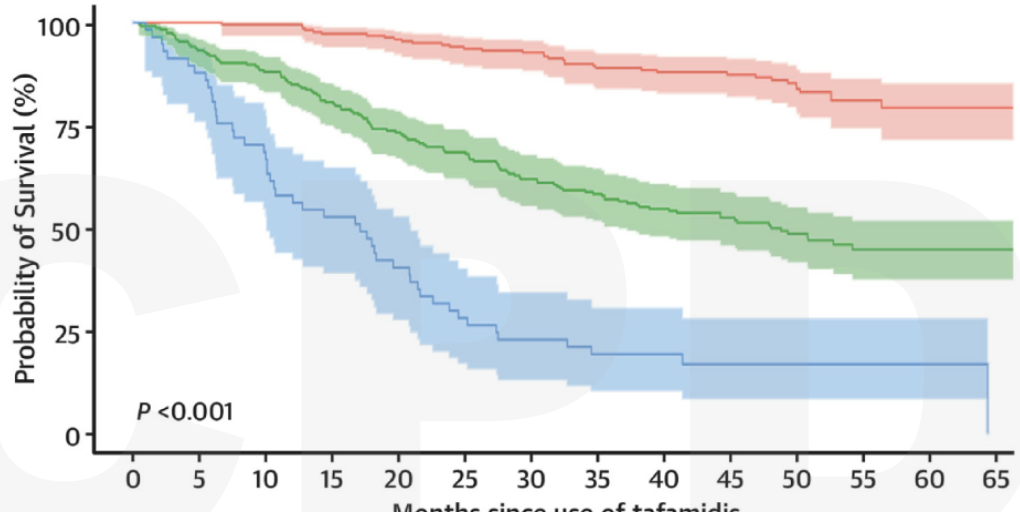


Median follow-up: 43.2 (Q1-Q3: 25.2-52.8) months
All-cause mortality: 38.6%

Probability of freedom from death at 65 months: 54.1%
(95% CI: 47.4-60.4)

Predictors of all-cause mortality:
Age, male, Black race, vATTR, Columbia Stage, time from diagnosis

Survival Secondary to Columbia Stage



No. at risk														
Columbia Stage I	218	218	217	212	208	203	201	188	171	142	114	58	35	4
Columbia Stage II	234	216	203	185	168	157	141	129	112	87	66	31	20	3
Columbia Stage III	57	50	38	30	23	16	13	11	9	6	6	5	2	0
Missing	115	113	109	106	101	94	91	84	73	58	43	25	21	0

Masri et al, 2023, JACC CardioOncol

Cardiac amyloidosis

- Suspect in cases of:
  - Unexplained LVH
  - HFpEF with LVH, especially in black population
  - Systemic symptoms: peripheral neuropathy, macroglossia
  - Low-voltage ECG unexplained or with TTE showing LVH
  - Apical sparing strain pattern on echocardiogram

- It is critical to rule out AL amyloid with serum FLC and SPEP
- PYP scan will confirm diagnosis of ATTR-CM
- There are effective treatments available to prevent disease progression

Case 3

HPI/CC: 65yo female with essential hypertension, T2DM, CKD III presents to clinic to establish care with chief complaint of shortness of breath and mild lower extremity edema.

Medications: insulin

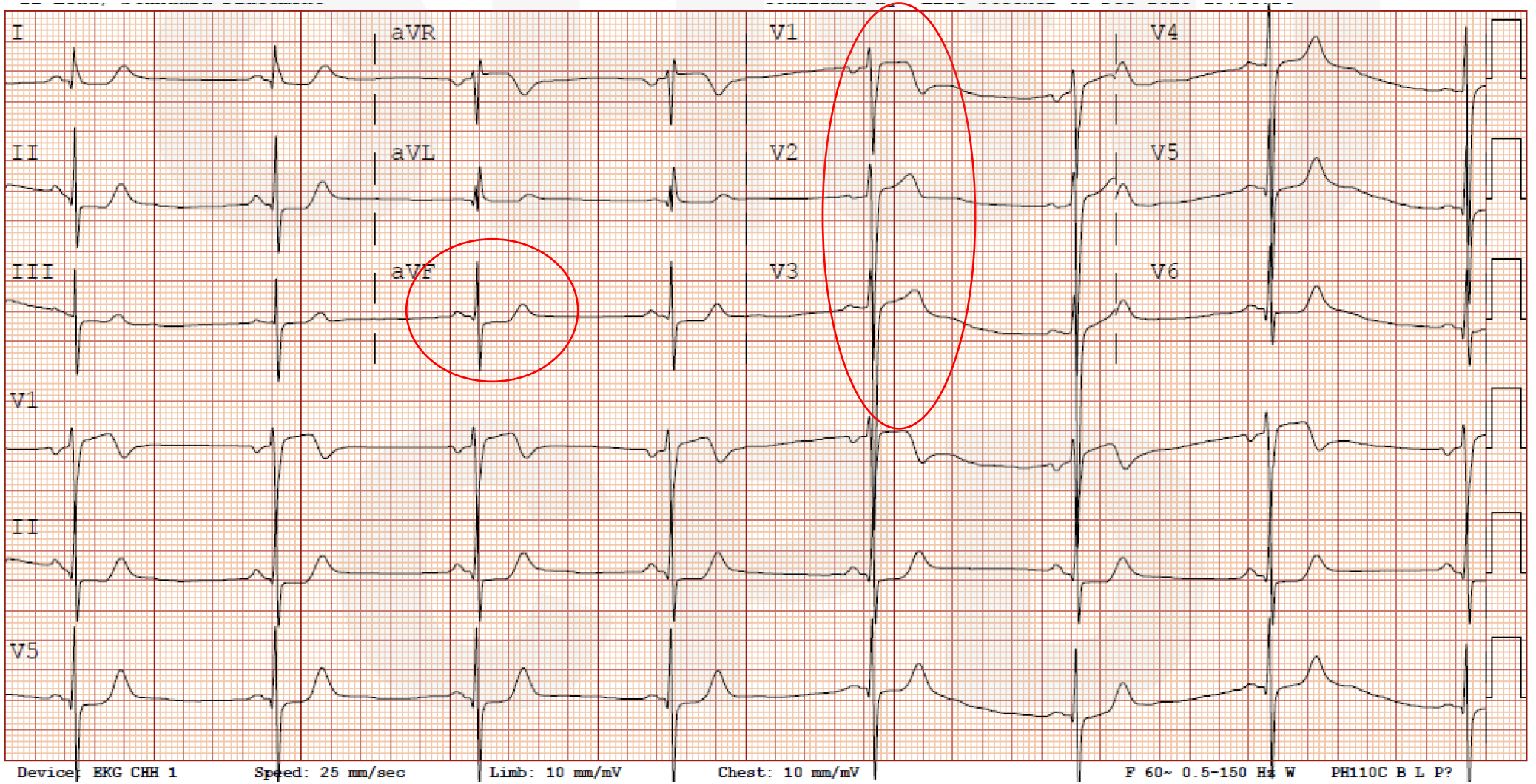
Allergies: None

Family history: father – died suddenly at 58, mother – MI age 67

Vitals: afebrile, BP 148/94, HR 60, 96% O2

Exam: RRR, no murmurs, JVP normal, trace peripheral edema

ECG

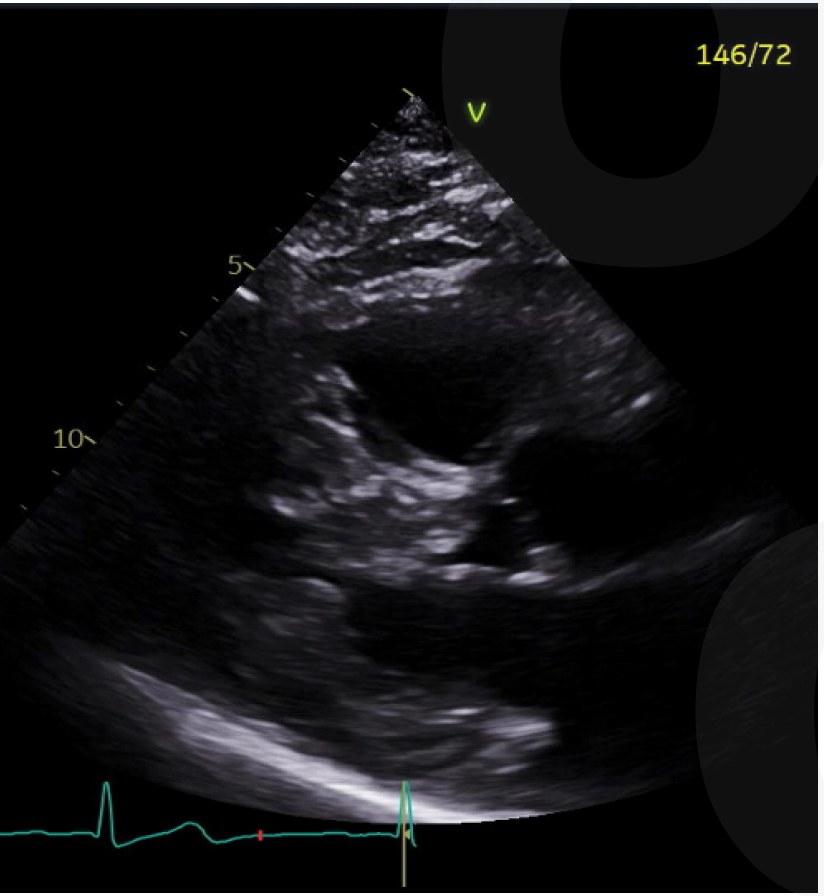

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
Device: EKG CHH 1
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
F 60~ 0.5-150 Hz W
PH110C B L P?

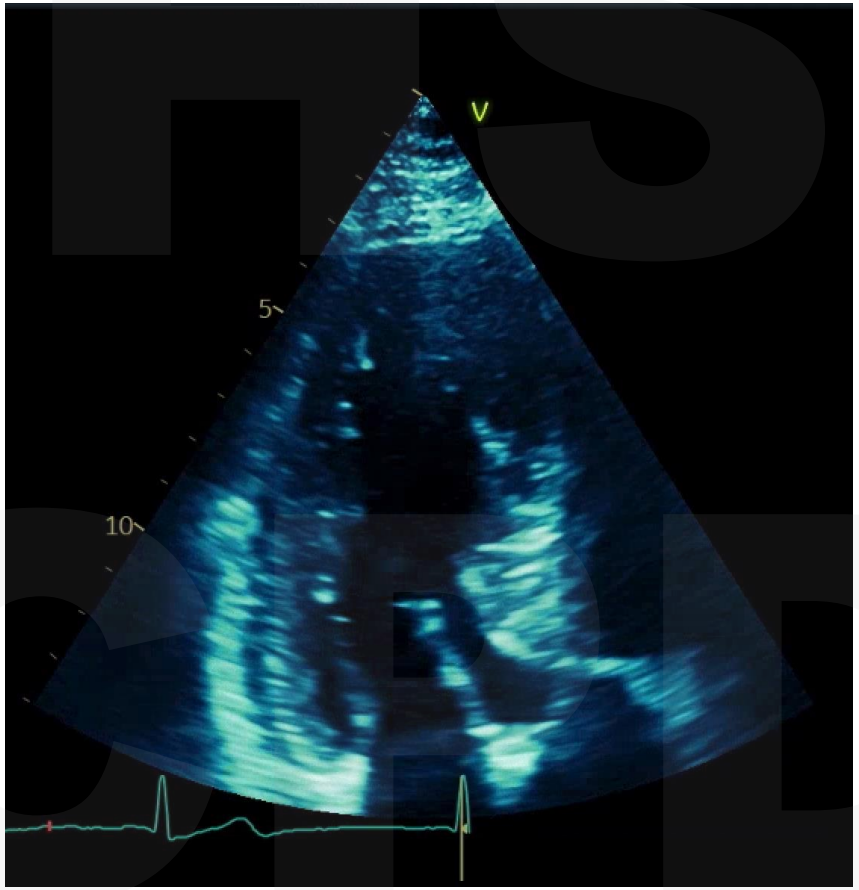
Echo report

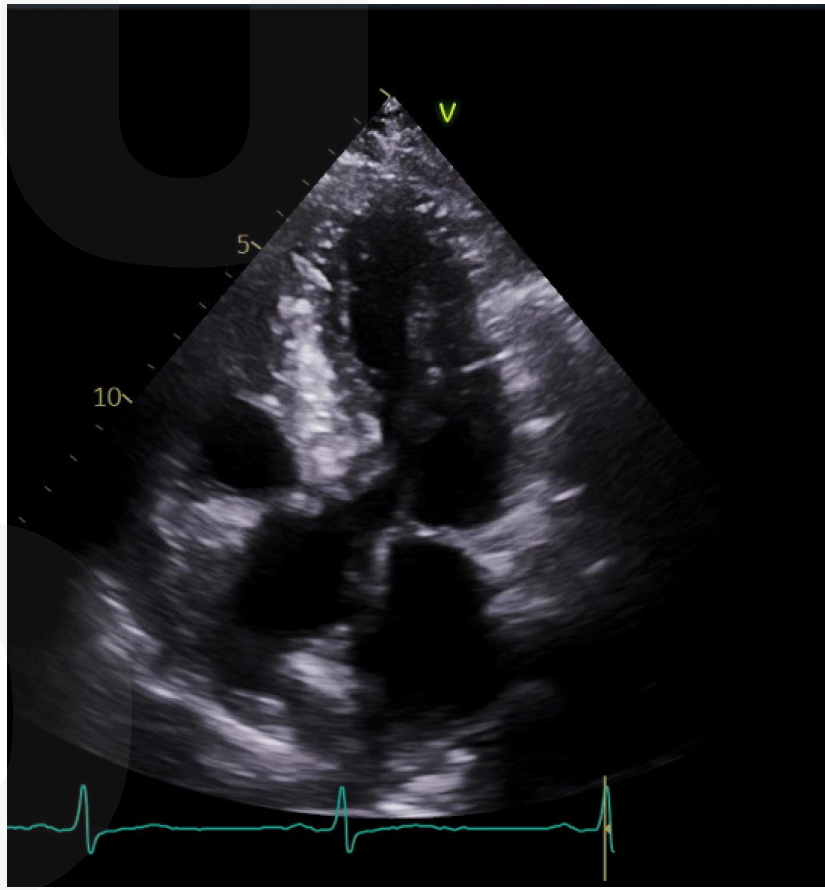
Impression:

- Left ventricular chamber dimension is small.
- There is moderate asymmetric septal hypertrophy (IVS 1.6 cm) with systolic anterior motion of the mitral valve.
- There is moderate LVOT obstruction.
- Left ventricular systolic function is hyperdynamic with a visually estimated EF of 75%.

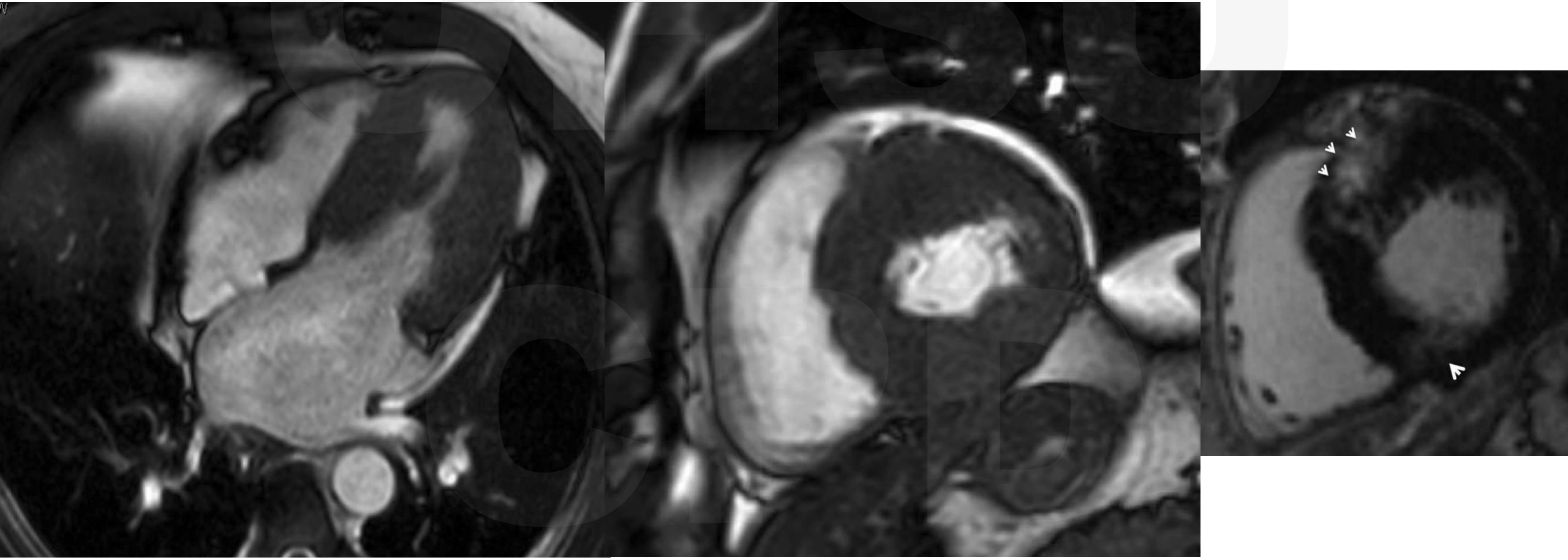
Echocardiogram







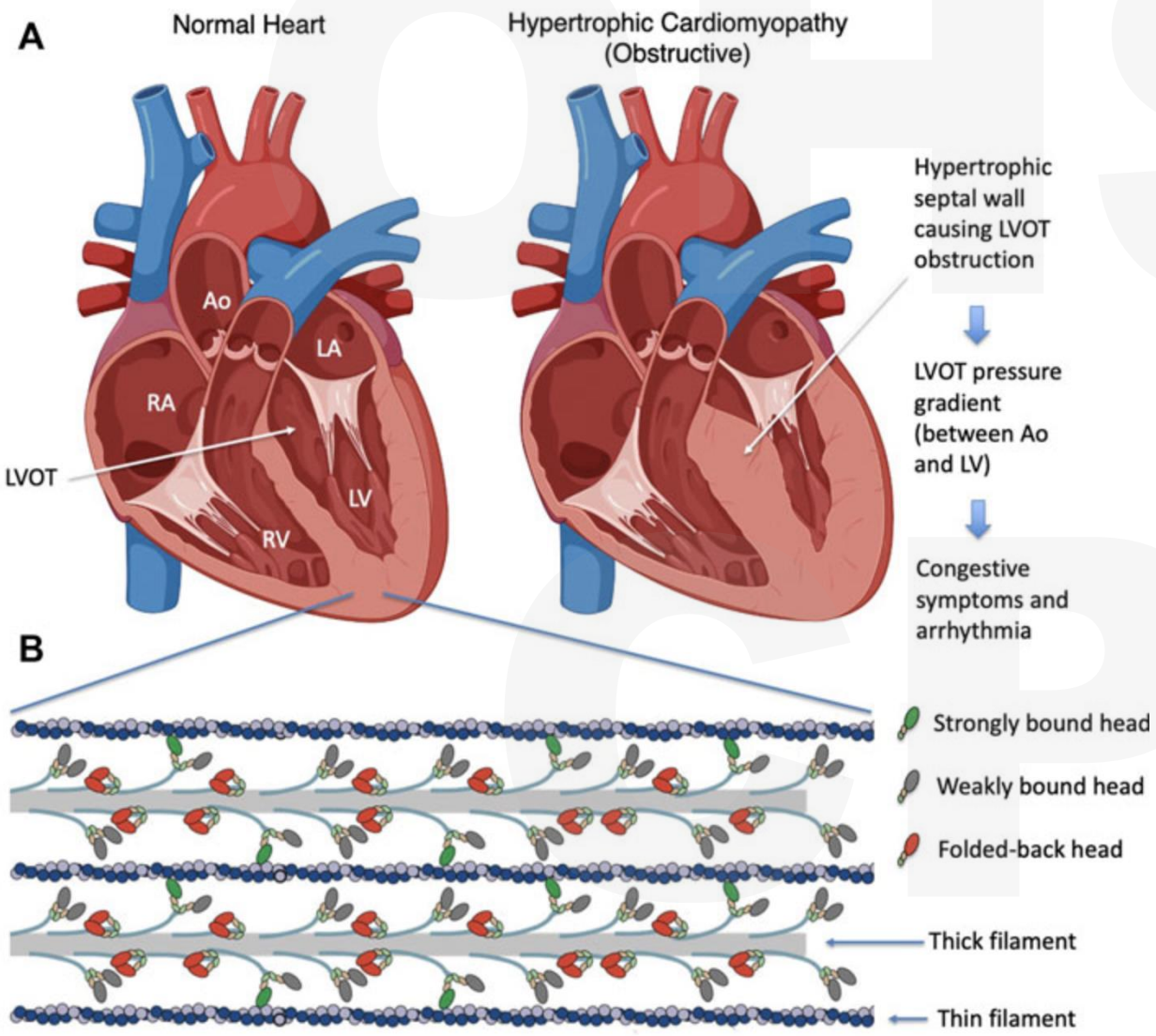
Cardiac MRI confirms asymmetric LVH and evalautes for fibrosis



Which of the following is a clinical manifestation of hypertrophic cardiomyopathy?

1. Peripheral neuropathy
2. Macroglossia
3. Spinal Stenosis
4. Sudden cardiac death ★

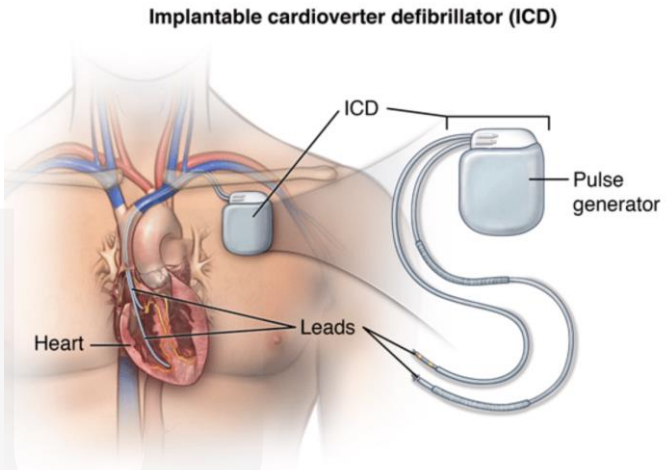
HCM Pathophysiology



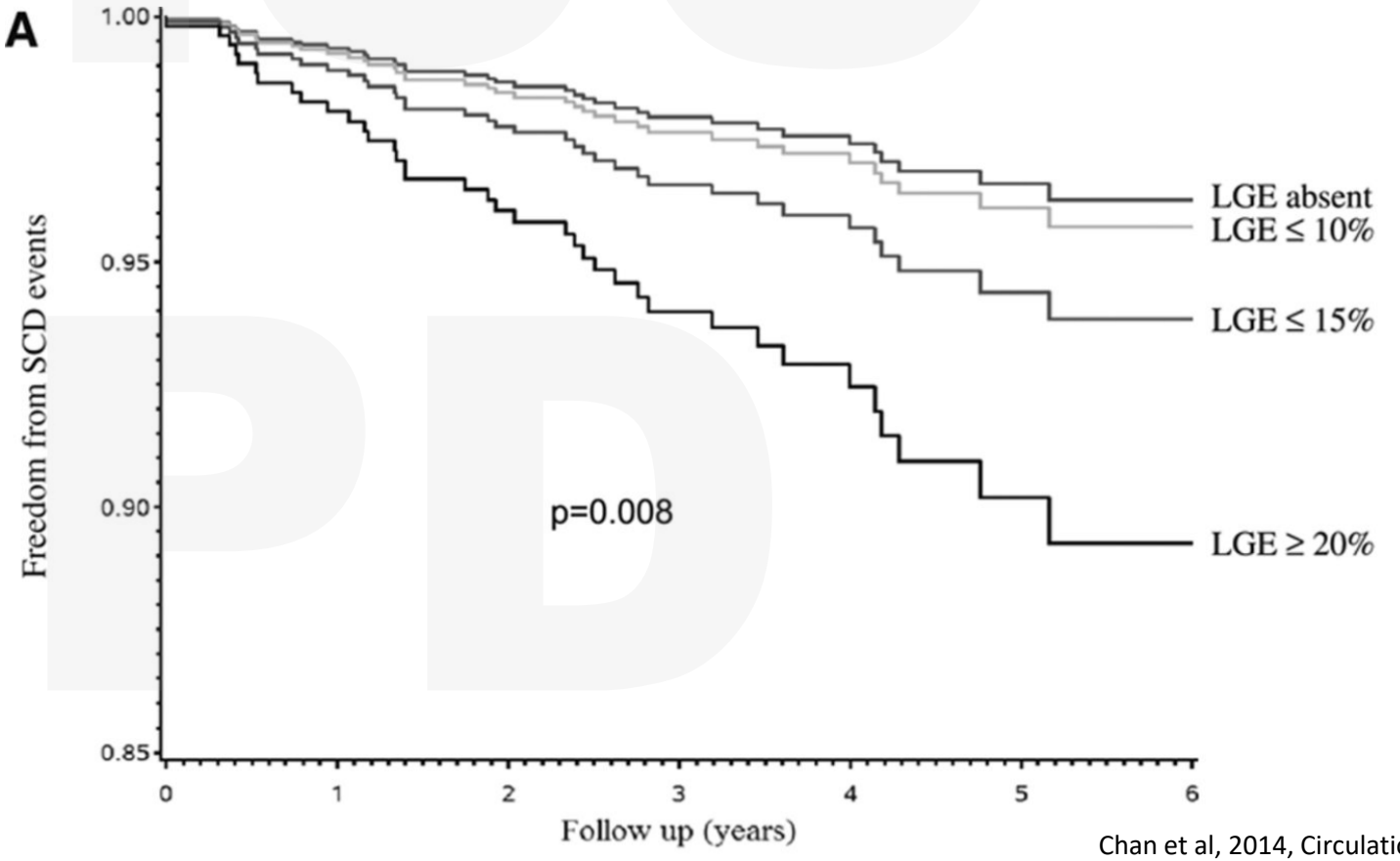
Heart Failure Symptoms

Risk of sudden cardiac death

Sudden Cardiac Death in HCM



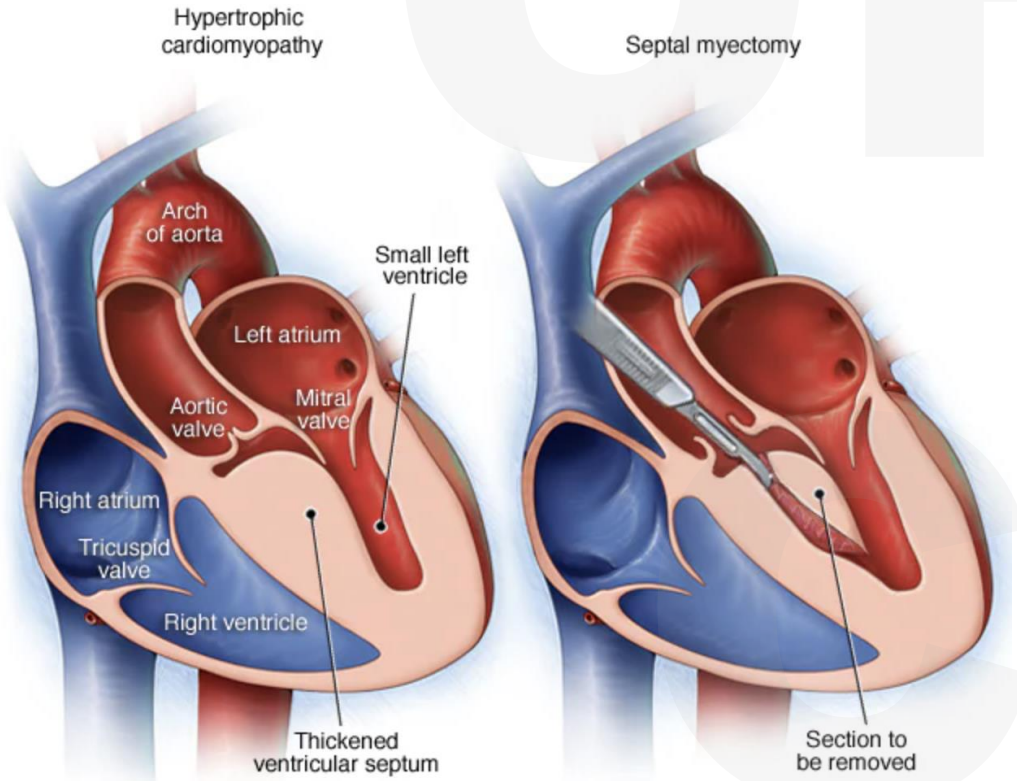
- LVH leads to fibrosis, increasing risk of lethal arrhythmias (VT/VF)
- Risk factors for SCD:
  - Fam Hx of SCD
  - NSVT
  - Unexplained syncope
  - EF <50%
  - Apical aneurysm
  - Extensive LGE
  - Wall thickness > 30mm



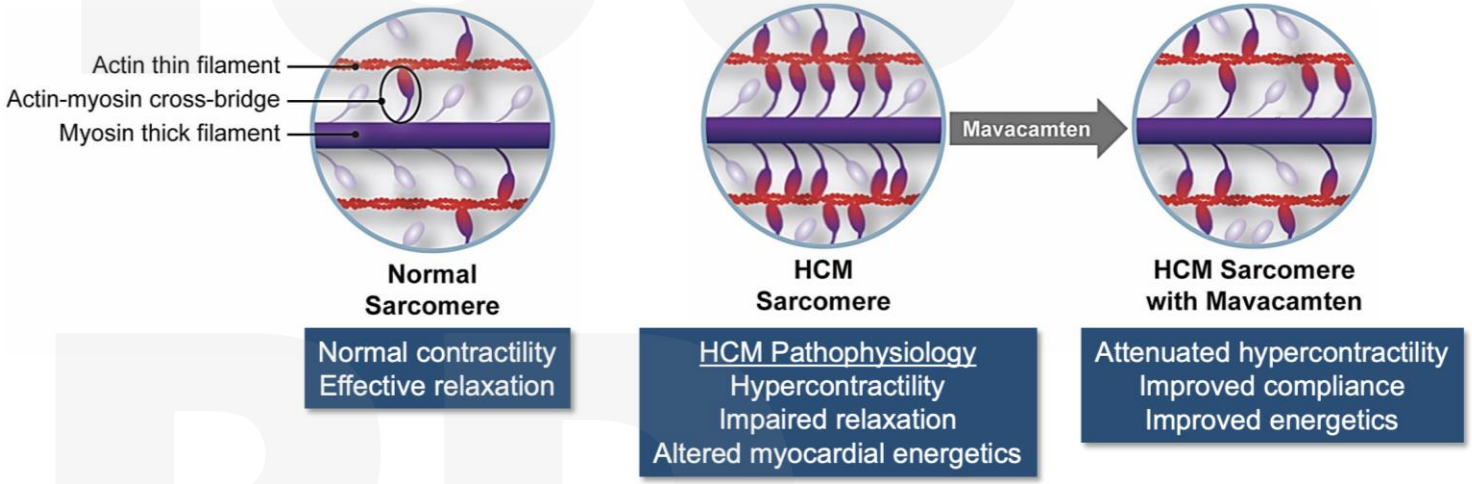
Chan et al, 2014, Circulation

Therapies for HoCM

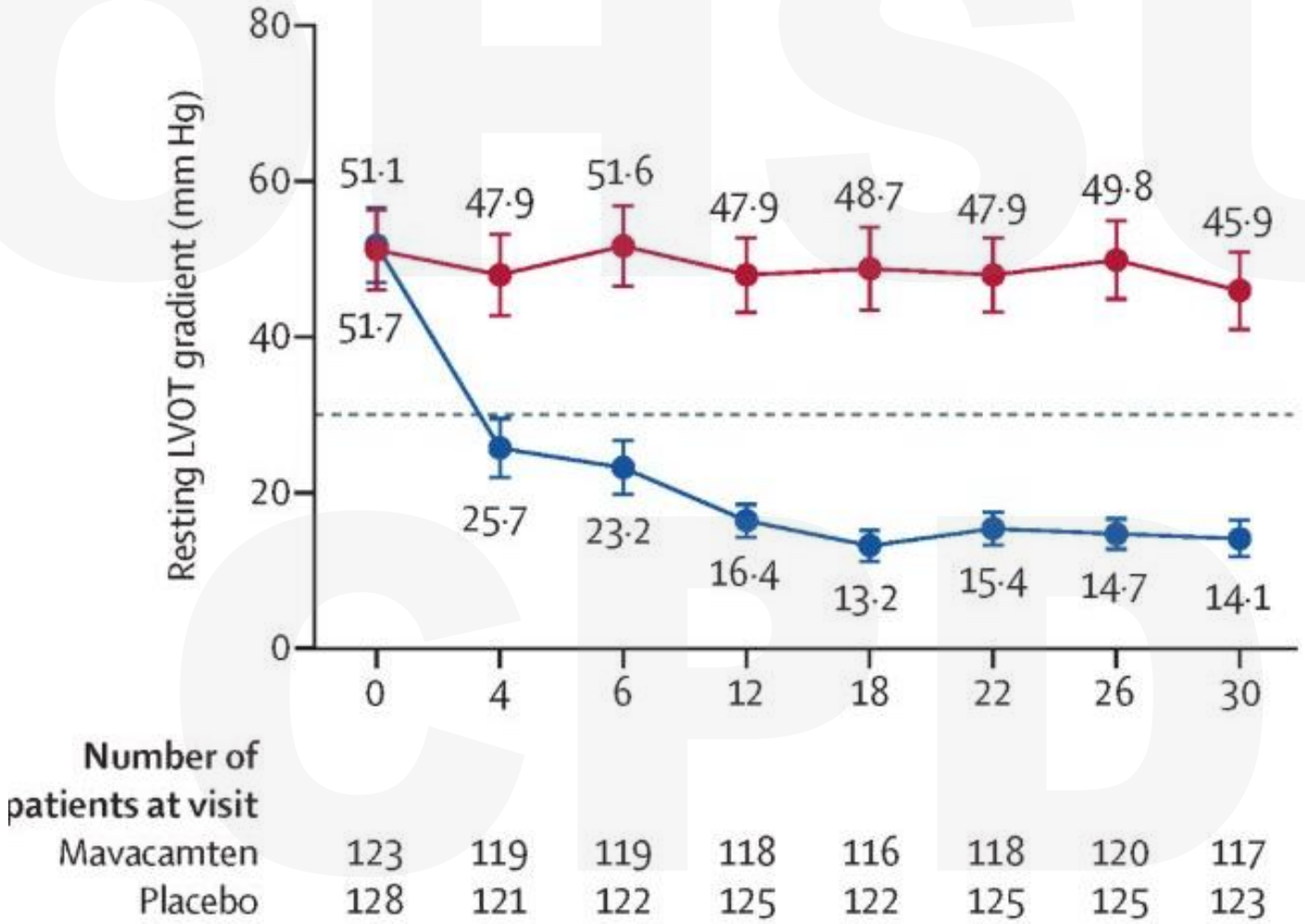
Septal Myectomy



Cardiac Myosin Inhibitors



EXPLORER-HCM Trial



Olivotto et al, 2020, Lancet

HCM Pearls

- Asymmetric LVH on echocardiogram should raise suspicion of HCM
- Family history is important part of this diagnosis (~50% are familial)
- Symptoms are generated primarily by presence of LVOT gradient
- Risk of SCD seems to be related to extent of LGE on cMRI
- Referral to cardiology is critical for evaluation of primary prevention ICD and disease modifying therapy

LVH Take Home Points

- Increased LV mass is associated with increased cardiovascular events
- Identification of etiology of LVH is critical to determining treatment
- Unexplained LVH should raise question of cardiac amyloidosis
- Asymmetric LVH on echocardiogram may suggest HCM
- There are now effective oral treatments for both cardiac amyloidosis and HCM

Questions?

- Thank you!

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